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# INTERNATIONAL CLINICS:

A QUARTERLY OF CLINICAL LECTURES

ON

MEDICINE, NEUROLOGY, SURGERY, GYNÆCOLOGY,
OBSTETRICS, OPHTHALMOLOGY,
LARYNGOLOGY, PHARYNGOLOGY, RHINOLOGY,
OTOLOGY, AND DERMATOLOGY,

*AND SPECIALLY PREPARED ARTICLES ON TREATMENT AND DRUGS.*

BY PROFESSORS AND LECTURERS IN THE LEADING
MEDICAL COLLEGES OF THE UNITED STATES,
GERMANY, AUSTRIA, FRANCE, GREAT
BRITAIN, AND CANADA.

EDITED BY

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# Drugs and Remedial Agents.

## SUPERHEATED AIR AS A HÆMOSTATIC.

BY DR. HOLLÄNDER,
Of Berlin, Germany.

The actual cautery has long been a favorite method for the destruction of pathological tissue in which were contained micro-organisms whose sphere of activity was liable to spread ever wider and wider unless their destruction *in situ* could be effected. It has also been the surgeon's most reliable means for controlling the parenchymatous bleeding that he was not able to stop by pressure and the tying of vessels. In both cases, however, it was open to serious objections which led to its non-employment until every other means had been tried. It destroys tissue so completely that it gives rise to unsightly and contractile scars that often are troublesome and inconvenient, and may even prove the site for the development of morphœa or even malignant neoplasms afterwards. Then, though pathological tissue is much less resistant, the actual cautery was not in the slightest degree selective in its action: all tissue that was touched, both the healthy and the unhealthy, perished.

This undesired excess in the action of the actual or Paquelin cautery is more or less necessitated by the fact that its action must be practically immediate upon the tissues which are naturally very rich in water, and so a high temperature of the iron must be employed. Besides this, the blackening of the tissues, superficially at least, at the immediate point of contact, the state of almost ebullition in the fluids of the part that follows the application of the hot iron, and the smoke of the seared masses disturb the appreciation as to the extent to which the cauterization has been carried. As the desideratum is

that it should be effectual, so as not to have to be repeated, at times more is done than is really needed.

What the surgeon and the therapeutist need is such a method of employing heat that the temperature can be regulated and controlled with nicety, and in which the result can be watched intelligibly and with proper appreciation of the amount of destruction of tissue that is being accomplished. Sometimes it is not desirable to carry the cauterization on to actual necrosis of tissue, but, if possible, to arrest it when micro-organisms have been destroyed and pathological tissue, owing to its lessened vital resistance, has been brought to such a condition that it will be thrown off, while surrounding healthy tissues may still react after the application of the heat and practically return to their normal state.

This, of course, is the ideal application of heat as demanded for external therapeutics, and it is this ideal that has been more or less perfectly reached by the employment of superheated air. A contrivance by which the superheated air is obtained is here shown. (Fig. 1.) It is simply an ordinary Bunsen burner, above which is fastened a coil of metal tubing, through which air is blown from an ordinary bulb or set of bulbs, as in the Paquelin cautery. The air is heated in passing through the coiled tubing, and issues from a nozzle so that it can be readily directed in any way that is desired. The heat of the coiled tube may be readily regulated by its appearance, from just under red heat to deep red, and the temperature of the heated air depends upon the extent to which this tube is kept heated. It is extremely easy to obtain from the apparatus a stream of air having a temperature of 300° C. (nearly 600° F.).

According to the size of the nozzle at the end of the tube, the air may be made to issue in any desired size of stream. As the heated air is rapidly cooled by contact with the atmosphere, the temperature of the effective stream of heated air may be regulated by the distance at which the nozzle is held from the tissues to be acted upon. The effect of the cauterization may be watched and its progress interrupted at any given point, only so much destruction of tissue being caused as seems suitable to the case.

For stopping parenchymatous bleeding the apparatus is extremely useful and easy to handle. Within the last three years I have had occasion to use it a number of times, and always with increased satisfaction. The most noteworthy occasion was after the removal of a

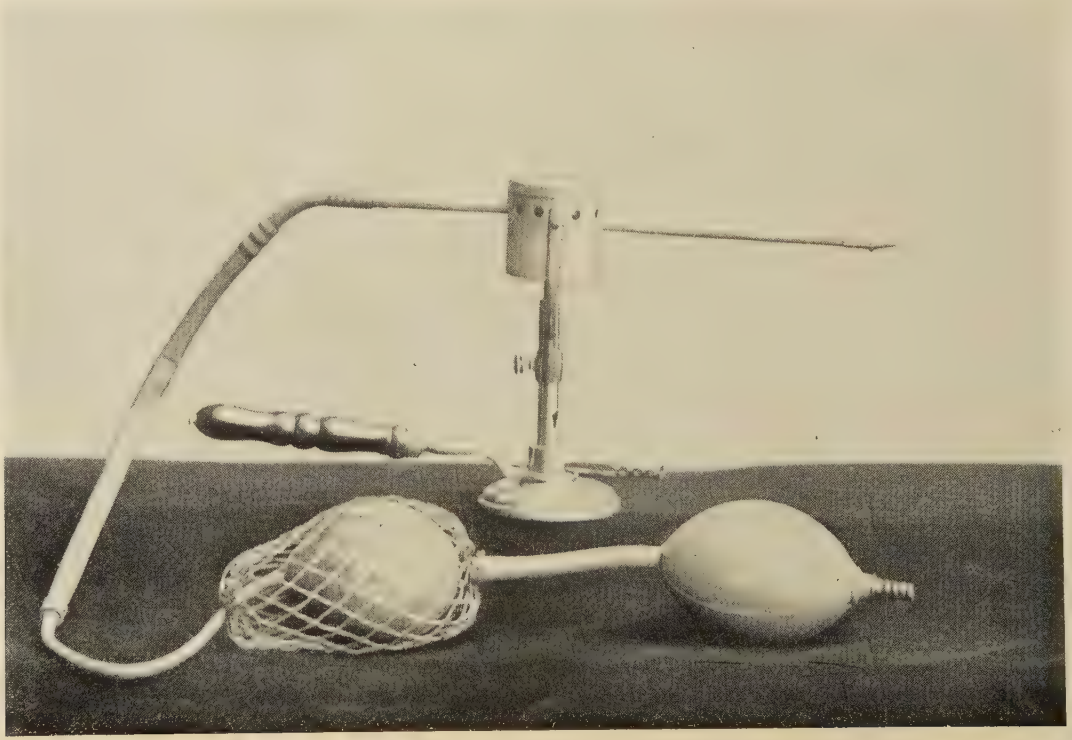

FIG. 1.—Dr. Holländer's apparatus for producing hot air.

cancer of the gall-bladder. The patient, a woman of forty-six, had some years before had a number of attacks of cholelithiasis. About three months before the operation she developed jaundice, which had persisted. In the right hypochondriac region, at the usual site of the fundus of the gall-bladder, a distinctly limited tumor was to be felt. It was very hard, and the distinction between it and the surrounding tissues was very marked. The patient had run down in health, with dyspeptic symptoms, seemingly from the absence of biliary secretion in the intestines. She begged very much for some operative interference that might ameliorate her condition, even though it involved some danger.

I felt that at least an exploratory operation was demanded by the condition. I found a cancer of the gall-bladder, which began very probably in the cystic duct, and which had already infiltrated the surrounding liver tissue. The cancerous infiltration seemed distinctly limited. No metastases could be noted in other parts of the liver as far as examination was possible, and the retroperitoneal glands were not demonstrably affected. The circumstances seemed extremely favorable for an attempt to extirpate the neoplasm completely by resecting the liver to what might be reasonably regarded as a safe distance beyond the limits of the carcinomatous infiltration.

The main difficulty encountered in such a resection is the stopping of the parenchymatous bleeding from the cut liver tissue. With the aid of the superheated-air apparatus I was able to accomplish it with very little loss of blood, an important consideration for the weakened patient, and with the production of only a superficial layer of cauterization of the liver substance. The subsequent course of the case was a most favorable one. In six weeks she was perfectly well and able to leave the hospital. Now, six months after the operation, she has no untoward symptoms, and there is every reason to hope that the extirpation was in the literal sense of the word a radical one and that my patient will not have a recurrence.

In the treatment of lupus vulgaris the use of superheated air has for some four years been of constant service. This disease is by no means rare, and, despite the number of methods suggested for its treatment by specialists of various classes, surgeons, physicians, and even bacteriologists, many cases obstinately persist or apparently get better only to relapse into a worse condition than before. Surgeons and dermatologists are not agreed as to which of their spe-

cialties it particularly belongs; as a matter of fact, however, in its earlier stages, those in which it is most amenable to treatment, it comes to the general practitioner. Some ready means that would be of service in the treatment even of the severer cases would be a welcome addition to the therapeutic armamentarium.

As lupus is now known to be the result of infection with the tubercle bacillus, the indication is for the removal of the infected focus in order to prevent further spread of the disease. For a time it was thought that the old tuberculin was going to prove a useful remedy in the treatment of this obstinate condition, but hopes thus raised were as sadly disappointed as were those that looked for a remedy for pulmonary tuberculosis in the new agent. The new tuberculin has proved no more effective, and its administration is attended with not less risk than that of the old, so that we are thrown back once more on surgical resources for the treatment of the disease.

Where the focus of infection is small and is so situated that its removal will not be followed by unsightly scarring, simple exsection is the manifest indication. But in the great majority of cases this is not possible, and it is in these that I have found the action of superheated air especially useful. The pathological tissue is less rich in blood-vessels, therefore less vitally resistant, and it contains less fluid and so is affected more by heat than healthy tissue; hence the hot air exercises a sort of selective action in its effect. While the diseased tissue is destroyed and thrown off, the healthy neighboring tissue is not affected so much but that it reacts and returns practically to its former condition, furnishing an excellent support for the cicatricial tissue that replaces the islets of lupoid tissue which previously existed, and supplying at the edges the epithelium whose overgrowth finally covers the area that has been cauterized.

The cases whose photographs are shown have been treated in this way. The difference between the appearance before and that after the cauterization is most striking. The sears are not too unsightly, not nearly so much so as in the cases where cure is sometimes spontaneously effected. To one who is accustomed to seeing the ravages made by lupus and the contractile scarring that results from the actual cautery or even from curetting, the results are very satisfactory. Attention is called especially to the fact that the scars thus produced do not pull upon and distort the eyelids and lips, as is so often the case after lupus.

FIG. 2.—Lupus of eighteen years' duration for which all the known methods of treatment had failed.

FIG. 3.—Condition two years after treatment. No relapse.

As regards the persistence of the cure after the treatment and the tendency to relapse so frequent in the disease, I have had in the three years since I perfected the technique of the method but one case of relapse. The case was that of a girl who had lupus not only of the nose and cheeks but also of the trunk and limbs. Her face was cauterized, with excellent results, perfectly smooth, white, and unsuspicious-looking cicatricial tissue replacing the lupoid spots. After some months, evidently from a new infection brought there from some other part of the body, she acquired a fresh lupous focus on the nose. Out of some fifteen cases treated this is the only relapse.

The technique of the method which has given such excellent results in my hands is comparatively simple. At first I used it in lighter forms of the disease and did not employ general anæsthesia. In severe cases of lupus, however, the pain of the cauterization would be too severe, and so general narcosis is necessary. The pain after the procedure is over is not severe,—in fact, as a rule, no complaint of discomfort is heard. I had a patient who complained very severely of a slight accidental burn on a healthy part of her arm, which occurred at the end of an operation, while the deep burn in the diseased tissues seemed to cause her no inconvenience.

As to complications of the treatment, there have been none. At first I hesitated to sear large patches on the face at a single sitting; but my hesitation has given place to assurance, as to the absence of any serious effects, after having seen more and more extensive parts subjected to the treatment, until now I know that the cauterization of a large part of the face, of the neck, and of the scalp gives rise to no troublesome after-symptoms. My fear that thrombosis of the superficial veins of the neck or some ulterior consequence of the heat on the skull or its contents might ensue has been entirely dispelled by the results of observation and experience. A temperature as high as 37.8° C. (100° F.) has been noted, but it seemed to be only a slight irritative fever, and was accompanied by no symptoms that would lead one to think of the absorption of even slight amounts of septic or toxic material.

Two degrees of cauterization are to be distinguished in the treatment,—a relative and an absolute, according to the extent to which it must be employed. These degrees of cauterization are separated by no hard-and-fast line, but one gradually merges into the other. Relative cauterization is a preliminary stage of the absolute cauter-

ization, which consists in a carbonizing or mummification of the affected tissues.

When the current of heated air acts on the skin, the epidermis rises up and the skin itself becomes extremely pale. This paleness is due to a sort of peristaltic contraction of the blood-vessels of the part, which causes a complete ischæmia or anæmia. This preliminary pallor is missed if the cauterization is done after a part has been emptied of blood by an Esmarch bandage. When the hot air is allowed to act further, the moist paleness of the skin gives place to yellow. The limits of relative cauterization are reached when this leather color takes on a dry shimmering appearance. The recognition of this period with exactness requires some clinical experience, but this is not difficult of acquirement.

This relative cauterization is the most important thing to be learned in the application of superheated air, as it is to it especially that the affected and unaffected parts of the skin react so differently. For this difference of reaction there is, besides the pathological reason that the affected parts have a diminished blood supply, a mechanical reason why the healthy tissue should be less influenced by it. When the heat acts upon tissue in the midst of which are tubercular nodules, the healthy tissue, by the peristaltic process that I have described, contracts and retreats farther from the source of heat, while the tuberculous material is forced forward and towards the heat. Slight distances at high temperatures make very great differences in the effect produced. The difference between the contractile properties of healthy and unhealthy tissue is in direct proportion to the extent of the affection, for in the tuberculous tissue the elastic fibres gradually disappear until practically none are left.

So it happens that, while the tubercles become necrotic and are thrown off, the healthy tissue recovers to a great extent after a few days, the circulation being restored almost completely. A proof of this selective action in the relative cauterization of the tissue is that the area acted upon is never thrown off in a single patch, but in smaller areas. In the cicatrization of a patch as large as the hand after cauterization, little islets of epidermal tissue occur all over the surface of the granulations, at points where the whole layer of epidermis was not destroyed, and from these as centres and by their confluence large superficial areas became rapidly covered with epidermis. So it is that after relative cauterization cicatricial contraction, and the

FIG. 4.—Lupus of thirty years' duration, most intractable to treatment.

FIG. 5.—Lupus of thirty years' duration one year after hot air treatment, absolutely without lupus nodules.

consequences for eyelids and lips that follow from it, do not occur, which is an invaluable advantage of the method.

The after-treatment is very simple. The cauterized parts must be carefully covered with lint on which boric lanolin has been spread, and then bandaged, using as much cotton as may be necessary to protect the parts from pressure and friction. The first bandage should remain on for three or four days at least. The œdematous swelling that occurs in the eyelids disappears after a day or two. The usual treatment for burns or for necrotic patches is then proceeded with, the surface of the granulations being freed as much as possible from necrotic particles. During the first few days the odor is often extremely unpleasant, but this disappears at the end of the first week, when all the necrotic tissue has been thrown off.

I have found it of advantage to paint the areas of granulation tissue lying between the islets of renascent epidermis with a three per cent. solution of nitrate of silver. It brings cicatrization about sooner, and yet adds no element of disfigurement to the resulting cicatrix. Wet bandages should not be employed. Usually cicatrization takes place rapidly. I have had but one case in which cicatrization was very slow, which was in an old lady who came with lupus that had existed for forty years. After a first absolute cicatrization, that proved a few days later, when the necrotic tissue had come away somewhat, not to have gone deep enough, I undertook a second one without waiting for cicatrization of the first. The result was extremely tardy cicatrization. It is well, then, in the old especially, to wait and see the result of a first cauterization before proceeding with a second.

Of some fifteen cases of lupus treated by this method I have had but one genuine relapse, that already mentioned, though some of the cases were cauterized more than two and one-half years ago and ten of them more than a year ago. I have been obliged in certain cases, not more than three or four altogether, in which the cauterization was done in patches of the size of the palm of the hand, to cauterize in an out-of-the-way corner here or there a nodule that seemed suspicious. To my mind, the same criterion holds for lupus as for malignant neoplasms, however, and we can only speak of curing when complete removal has been accomplished and the patient remains for three years free from any relapse. So I shall as yet only claim for my patients a noteworthy improvement.

I have used this method of treating lupus now for more than two and one-half years, and confidently believe that others who give it a fair trial will find, as I have done, that it is an important addition to our therapeutic armamentarium for the treatment of one of the most obstinate yet conspicuous and deforming skin diseases that we have to deal with.

FIG. 6.—Lupus of eight years' continuous duration.

FIG. 7.—Condition after the second application of hot air by the Holländer method.

# HOW TO GIVE DIGITALIS IN ORGANIC HEART TROUBLE.

CLINICAL LECTURE DELIVERED AT THE PITIÉ HOSPITAL.

BY S. JACCOUD, M.D.,

Professor in the Paris Faculty of Medicine.

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Gentlemen,—I have on several occasions in our wards called your attention to a patient whose history is of great interest, both on account of the organic lesions he presents and of the therapeutic indications to be drawn from them.

This man, sixty-eight years of age, robust, a butcher by trade, came to our wards with all of the usual exterior signs of organic heart disease. He showed marked oppression in breathing and œdema of the legs, both of which were explained by the condition of the pulse and heart. The pulse was wildly irregular and the heart-beats also were unequal, hurried, and confused. This condition of the heart having been noted, we were authorized to attribute the disordered peripheral circulation to the functional insufficiency of that organ; as whenever you meet with this inequality and irregularity of the cardiac rhythm, combined with oppression and œdema of the legs, you will have a right to infer that the heart is not up to its work.

After looking for the stethoscopic signs we failed to find an explanation or cause for this functional insufficiency. There was a slight systolic murmur at the apex propagated a little towards the axilla, which denoted a slight degree of mitral incompetence; but that was all, and we could find no other abnormal sound.

This amount of mitral incompetence could not account for the cardiac disorder and the other symptoms. You must not imagine that in cardiac pathology when you have found and localized a murmur you can deduct your diagnosis from it by mathematical equa-

tion. This may be true in certain cases, but it is the exception. With our patient it was not possible to make a comparative inference from the results of auscultation and the symptoms present. In the absence of any hepatic, pulmonary, or renal complications (there was no albumen in the urine), we were not warranted in saying that his mitral incompetence was the sole cause of the circulatory disorder present.

But there is a disorder of the heart that may have no other sign than irregularity; I refer to myocarditis. This disorder may exist without any murmur at all, and could account far better than the mitral incompetence of our patient for the functional insufficiency he presents. On general principles it is preferable to have a well-compensated mitral incompetence than myocarditis.

Consequently, with our patient we made the diagnosis of chronic myocarditis with slight mitral incompetence, and, as he was in a marked condition of cardiac insufficiency, it was our duty to come to his assistance as rapidly as we knew how. This urgency was all the more marked since, in addition to the signs I have mentioned, there was already an alarming decrease in the urinary secretion.

I began by prescribing a milk diet for twenty-four hours, and on the following day an infusion of eight grains of powdered digitalis leaves. The result was most satisfactory; under the influence of this moderate dose of digitalis, the way having been prepared by the milk diet, remarkable improvement was noted in twenty-four hours: the oppression had diminished, as well as the œdema of the legs, the irregular heart action was less marked, and the kidneys had secreted freely (a little less than three quarts).

When I saw this, I followed my customary habit and prescribed for the following day a smaller dose of digitalis infusion,—five grains, instead of eight.

The next morning the scene had completely changed. Relapse to the original condition had been as rapid as his improvement; in fact, our patient was, if anything, worse than when he entered our wards. The diuresis had fallen below the normal again, the patient was anxious, the irregularity and particularly the frequency of the heart-beats, which had been specially influenced by the first dose of digitalis, had reappeared, and, finally, there was bilious vomiting. The contrast between the patient's actual condition and that of the previous day was therefore most striking, and we had returned to

the point from which we had started, with an additional symptom, —the vomiting.

The interpretation of this change was a very important and not an easy matter. The first thing to be done was to find out whether we had given too much digitalis or not enough.

You must not think that it is for the pleasure of arguing that I raise this question; the way in which I just put it corresponds exactly to what you will have to do in practice. You must always bear in mind the relative action of drugs, and, even when you have given small doses, keep an eye on the way they are acting. It is not altogether the size of the dose that is important; you have to consider your patient's individuality as well: a dose that will not be enough for one patient will poison another one! In order, therefore, to carry a treatment through skilfully you must consider your patient, and not the doses of the remedies you administer.

In circumstances of this sort you have to decide the matter on the spot. Evidently we had not given too much digitalis, if we look at the size of the dose alone, but it was possible that we had given too much for this patient's idiosyncrasy. To decide this point you must be familiar with the signs of digitalis poisoning. With our present patient I followed, as I said, my usual method of administering digitalis, and yet at the very start I was brought up short by evident intolerance for the drug, manifested by signs that every one ought to know. These signs are numerous, and when they all appear in a patient you cannot fail to recognize them.

It is widely believed that the first signs of intolerance are necessarily nausea and vomiting. This may be so, and no doubt frequently is so, and the physician is considerably helped in interpreting the symptoms when he is told by his patient that he has vomited. This simplifies the question, but the situation is not always so clear as this. It sometimes happens that the earliest indications of intolerance completely escape the patient's notice, and are only perceived by the physician who takes the trouble to look out for them.

To begin with, there is the state of the pulse, a quite special condition, which has no resemblance to the condition at the beginning of the treatment, when it is strong, slow, and regular. Instead of being slow and full, when there is intolerance for the drug it becomes just the opposite,—quick and weak. This very definite change in

the condition of the pulse may, chronologically, be the first sign of intolerance.

On the other hand, it is also possible that the first symptom that manifests intolerance may be the decreased diuresis; this is perhaps as common as to have the scene open with a change in the pulse. Just when you have congratulated yourself on the free diuresis produced by the action of your remedy, you suddenly see the quantity of urine fall back again to its original point. This is what occurred with our patient. Therefore, in wishing to detect the first signs of intolerance do not rely altogether on the pulse, but when you see the diuresis falling off you need wait for nothing farther and can take your precautions without further delay. If you wait until vomiting sets in, you may make serious mistakes, as vomiting may appear only in a late stage of the case.

Nor is this all, as there are cases more deceptive still, though, fortunately, they are less frequent. There is sometimes at the beginning a phase of intoxication manifesting itself by cerebral activity, insomnia, and various sorts of nervous phenomena; these are afterwards followed by the usual signs to which I have referred above. You will do well to bear in mind the possibility of this occurrence.

These points being understood, you will easily see that, when I saw in our patient the presence of all of these signs except the nervous symptoms, I readily detected the intoxication by digitalis, in spite of the moderate dose that had been used, about thirteen grains of infusion of digitalis in the space of forty-eight hours. Once more let me say that figures are of little importance in occurrences of this sort when the phenomena succeed each other so rapidly. Cases are not infrequent where accidents due to intoleration appear inside of five or six days; the physician, who fancies his patient has become accustomed to the action of the drug by that time, is not on the lookout for intolerance and is taken off his guard. Such disasters may end in asystolia and death. But by paying ordinary attention and by not being guided too much by figures and doses, such an error ought not to occur. Never think you are safe from any risk of intoxication because you are giving small doses; bear in mind the duration of your treatment and the well-known accumulative property of digitalis.

When you find yourself in the presence of signs of intoxication,

your line of action is not a difficult one. You must suppress the digitalis at once; but, as the indication for the administration of such a drug still exists, you must fall back on another remedy that will be able to a certain degree to take the place of the digitalis which you are obliged to suspend for the time being. Such a remedy you will find in caffeine, which has its drawbacks also, but they are not those of digitalis. I gave our patient fifteen grains of caffeine, and the result quite equalled my expectation, as in twenty-four hours the diuresis increased and the irregularity in the heart action ceased. In forty-eight hours we had regained all the ground lost, and the patient's condition was again as hopeful as when he had taken his first dose of digitalis.

I did not limit myself entirely to this dose of caffeine, but gave him two ounces of lactose as well,—a diuretic of no mean value.

I have during this lecture spoken several times of the infusion of digitalis, which is the form in which I always give this remedy. My experience, which is now a long one, has convinced me that this is the best method of administering it. In some cases, however, I use digitalin (though never amorphous digitalin),—for example, with patients who will do best on small doses continued for some length of time; but in urgent cases, such as ours, where there is no time to lose, I always resort to the infusion of powdered digitalis leaves. You should not use powder that has been prepared in advance; the leaves should be crushed for the prescription, boiling water poured on and allowed to stand twenty minutes, the preparation filtered (which is too often neglected), and sweetened with an ounce of simple syrup or of syrup of bitter orange bark.

The dose of powdered digitalis leaves employed in this way can vary between three and fifteen grains in twenty-four hours; the latter dose I regard as a maximum, beyond which it is not advisable to go.

The method which I generally follow in the administration of digitalis prepared in this way is as follows. On the first day I give the maximum dose that I think the case requires,—from eight to fifteen grains. With digitalis I do not use the steadily increasing doses that you see me give of certain other drugs. This maximum dose, however, I give only once. The next day I lessen it to a varying amount according to the effect produced. The third day I lessen again, unless a proper result has not been obtained, when I repeat

the dose of the second day. Never do I use the drug for more than five consecutive days; when this has been done I let the patient rest for three days, and then, if necessary, begin with the digitalis again. It was in accordance with my rule that on the second day I gave our patient five grains, and it was fortunate for him that I reduced it so much, as, if I had given him a second dose as large as the first one, the resulting intoxication would have been much more serious.

If you handle the drug in this way and are thoroughly posted on the signs of intolerance and their chronological sequence, you will never have any trouble.

There is still another point that I must mention. This form of intolerance, such as we saw in our patient, is a secondary one; but you must know that there is a form that is primitive. There are some persons who cannot take the slightest amount of digitalis without vomiting; they show their intolerance from the outset. With such patients you must use caffeine.

There are three ways of giving caffeine. Its dose varies between four and eighteen grains. It can be given in a wafer, as a first method; in solution, as a second, but then it must be combined with an equal amount of benzoate of soda to get it to dissolve; thirdly, in hypodermic injections, when there is gastric intolerance. There are many formulæ for these injections, but the main point for you to settle is the quantity given per syringe. A useful solution is one of equal parts of caffeine and benzoate of soda in water, of such strength that each injection will contain six grains of caffeine.

But you must also know that caffeine cannot be given to all patients for any length of time. With this drug also you will get intoxication in some cases, manifesting itself by insomnia and agitation, and you will then have to cease its use.

# Treatment.

## ON THE PREVENTIVE TREATMENT OF PATERNAL SYPHILITIC HEREDITY DURING PREGNANCY.

CLINICAL LECTURE DELIVERED AT ST. LOUIS HOSPITAL.

BY ALFRED FOURNIER, M.D.,

Professor of Dermatology at the Paris Faculty of Medicine.

Gentlemen,—It is my habit, at the beginning of the semester, to devote my first lecture to a practical subject that only presents itself exceptionally in hospital work, but is, on the other hand, of frequent occurrence in private practice. It is therefore my intention to-day to treat the following question: At the beginning of a pregnancy in a family where the husband is syphilitic but the wife healthy, the syphilis of the husband being at a stage where it may be hereditarily dangerous to the child, can we by treating the mother protect the child?

This means that we are to give the specific treatment to a woman who is, as regards syphilis, perfectly healthy; who has never presented the slightest symptom of syphilis; and who is, therefore, merely to play the part of a filter through which the specific remedies are to pass to reach the child who stands in need of them.

Let us see in what manner this problem presents itself in the course of daily practice.

All these cases belong to one or the other of two classes: it is either the first pregnancy in the family, or else there have already been several which have ended badly.

In the first class the occurrence is generally as follows. A gentleman comes to your office and tells you that he has not come for

himself, as he, fortunately, is in good health; but that he was recently married, and finds that his wife is *enceinte*. This fact would only give him pleasure were it not for a dark cloud on the horizon: he had syphilis at such and such a date, and has heard that calamity awaits the children of syphilitic fathers,—that they either die before birth or are born in a diseased condition. He, therefore, wants to know whether anything can be done to prevent the child's inheriting the disease.

Let me here make a remark in parenthesis. Do not think that this situation is a more or less rare event. It is an absolutely common occurrence, that you will meet with many a time in your practice. My experience has led me to form the following axiom: however indifferent a man may be to his syphilis before his marriage, he takes a very different view of it as soon as he sees the prospect of his becoming a father, and this is a very curious psychological phenomenon. He may not give a thought to his syphilis when he wants to get married, but as soon as he finds his wife *enceinte* he is a different person, and is haunted by the remembrance of his disease: What will be its effect on the child? Will it betray him to the world, or give the disease to his wife? etc.

The second class of cases comprises those in which in a family several pregnancies have ended badly, the wife is *enceinte* again, and it is concerning this new pregnancy that your advice is desired. Such cases are similar to the following one, which came under my notice only a short time ago.

A perfect stranger came to my office and told the following tale: "I have been married for several years and my wife is just beginning her fifth pregnancy. The others have all ended in disaster, the first three by unexplained miscarriages, and the fourth by worse still: my wife had hydramnios, the baby was hydrocephalic, and its head had to be crushed. A well-known obstetrician whom our physician called in for this operation took me aside and asked me whether I was not syphilitic. I was, but denied it to him. But his reply was, 'Never mind; take a mercurial treatment if you want to have children.' I did not do so, as I had fully made up my mind to have no more children. In spite of this resolution my wife is again *enceinte*, and I have come to you to know whether there is any way of avoiding a fifth catastrophe."

Such are the two forms in which the situation will be laid before

you. It is my duty to show you that this protection of the child to be born, that these fathers desire at our hands, can be furnished to them by giving the specific treatment to the fœtus through the mother, although she is perfectly healthy; and I desire to show that this mode of procedure is rational, free from all risk to the mother, and efficacious as regards the fœtus.

It is rational in that it brings the specific treatment in contact with the fœtus,—the patient,—and that without doubt. Experiments on the power of the placenta of absorbing drugs have shown that potassium iodide given to the mother appears in the fœtus's urine in forty minutes. Other experiments have demonstrated the presence of mercury in the bodies of fœtuses whose mothers had followed a mercurial treatment.

It is devoid of all risk to the mothers, as experience has shown that non-syphilitic women tolerate the specific treatment just as well as syphilitic ones; that the treatment does not create gastric disorder, nor increase such digestive troubles as commonly occur in pregnancy; and that it does not add its anæmic effect to the anæmia attendant on pregnancy. I have yet to see a case in which the specific treatment properly administered has been the source of any trouble to a pregnant woman, and my colleague, Professor Pinard, is of precisely the same opinion.

As regards its efficacy for the fœtus, there is no shadow of a doubt. It does not save them all, but it protects the great majority of them. When the treatment is given to a healthy woman *enceinte* for the first time (the husband being supposed to be syphilitic and at a period of possible or probable danger to the fœtus), and is begun at the beginning of pregnancy, the latter usually goes to full term, and the child is born alive and generally healthy. If the treatment is given when pregnancy is already well on, it has many chances of failing. It is, of course, possible that a healthy child would have been born without any treatment; cases of this sort do not absolutely prove the efficacy of the method, as do the following group:

When, in a family where several pregnancies have already ended in disaster, at a new pregnancy the mother, who has heretofore received no treatment, is put on the specific remedies (the case being always as above: mother healthy, father syphilitic), the child can be born alive and healthy. Such cases, which are relatively common,—for my part, I could show about fifty such,—are a mathematical

demonstration of the value of the method for the fœtus, or else there are no such things as logic and common sense.

Not only can an abrupt stop be put in this way to a series of miscarriages in a syphilitic family, but the specific treatment given in such circumstances may eliminate a certain number of pathological phenomena that had occurred during previous pregnancies. Thus, I have cases that show that hydramnios, diseased placenta, fœtal dystrophy, and particularly hydrocephalus, can be avoided by the specific treatment.

It is a curious and rare fact that as regards this question of special therapeutics there is almost unanimity between obstetricians and syphilographers; all of the leading accoucheurs of Paris, with whom I have taken occasion to speak on the subject, hold exactly the same opinion as I do on the matter.

It must not be inferred from what has been said that I advise the specific treatment in *all* cases where the father is syphilitic. It is self-evident that no such general rule can be laid down, but that each case must be considered by itself. The necessity for this indirect treatment of the fœtus is obvious when the father is at a period of the disease where he constitutes a danger for his offspring; but no one would think of adopting such a course if the disease has become manifestly inoffensive through duration, long treatment, a period of immunity, or the birth of living and healthy children.

In these two instances there is no room for doubt, but between these extremes there are a multitude of cases where the problem is not so simple. When in such a dilemma there seem to be as many arguments for as against, give the specific treatment. If you do not, the fœtus may suffer harm; if you do, all you risk is to have done something that is of no use.

If we suppose, then, that you have decided to advise a specific treatment, you will still not have solved all the difficulties of the situation. The mother to whom you are going to give this treatment may or may not know that her husband has had syphilis.

When, for one reason or another, she is already in possession of the facts of the case, you will meet with no opposition, particularly when you have explained to her that the treatment is for the welfare of the infant. On this point you may accept my word for it,—never, either in private or in hospital practice, have I seen a mother refuse such advice, provided it had been made clear to her that it was for

the good of the child. This, however, is not the usual situation in which you will find yourself; it is much more frequent that the wife is ignorant of the whole affair! You have then, in spite of the fact that she thinks she is in a perfect state of health, to persuade her to follow a course of treatment, and to give her a mercurial treatment.

There are two ways, naturally, of going about this. One is to own up frankly, in which case the situation is reduced to the same terms as the preceding one. The other is to proceed in such a way that she shall not suspect the reason why she is being treated nor what remedy is being given to her.

The choice between these two methods concerns the husband, evidently, and not the physician; and, as a rule, the husband asks you to treat his wife, if nothing else can be done, but to do so without betraying him. Now, as we are obliged to do as he wishes, we find ourselves in a position of complicity and deceit that is anything but agreeable. You have to invent a pretext for giving any treatment at all, and have to disguise your mercurial pills under any euphemism that may seem to you most likely to pass under the circumstances. Then, if your patient is of an inquiring turn of mind and undertakes to question you a little about the whole affair, you may be driven into actual lying to hold your ground. So you see that the medical man in such a case is placed in a thoroughly false position, to which the finishing touch can be put by the wife's mother, if she happens to have a tendency to mischief. In one instance where my late colleague, Professor Tarnier, and I had, in order to save a husband, positively denied that the pills we were giving contained any mercury, the mother-in-law had them analyzed by a chemist, and, armed with his report, came to us and put us through a very unpleasant quarter of an hour!

Still, in spite of this unpleasant side of the question, you must act as I have advised. It is evident that you are in the right, that your office is a beneficent one, and that, after all, the dilemma is none of *your* making. Even if you do get caught, the situation usually straightens itself out. Thus, when Tarnier and I again met at the house of the patient whose mother had been our undoing, so far from meeting with reproaches, she shook hands with us affectionately, and told us not only that she was not angry with us, but that she wished to thank us for what we had been quite right in doing.

We now come to the question of treatment. An indispensable

element of success is to begin it at as early a date as possible. When the mercury is administered from the beginning of pregnancy, it will often be successful; the later it is given the less will be your chance of success; if it is not given until after the fifth month, it is too late and there will be but little hope.

Mercury is the best remedy. It is far superior to potassium iodide as a preventive remedy and as a corrective agent to hereditary taint. That iodide of potassium should be combined with mercury, or given *between* periods of mercurial treatment, can only be advantageous; but do not forget that your mercury is the *sine qua non*, that it is indispensable, and that success depends on *it*.

Anyhow, it is vastly easier to get your lady patients to take mercury than potassium iodide, and you must not forget to take advantage of everything that will tend to make the treatment as little disagreeable as possible. The potash salt will give your patient coryza, acne, a metallic taste in the mouth and bad breath in the morning, and she will not thank you for any of these manifestations.

As regards the form in which you are to give the mercury, you have not much choice. Frictions and injections would probably betray you very quickly, when the mother does not know what is being done; so that you must give it by the mouth. The proto-iodide in pills has seemed to me to be the preferable form. It is an active preparation, easy to take, and well tolerated by the digestive tract. It is better than sublimate, I think, as the latter irritates the stomach, and when given in solution soon disgusts the patients by its horrid taste.

Professor Pinard, on the other hand, has adopted the following preparation, which he gives either as a solution or syrup:

℞ Biniodide of mercury, 10 centigrammes (1½ gr.);
Iodide of potassium, 10 grammes (150 grs.);
Water, or syrup of sugar, 250 grammes (8 oz.);
Mint water, 50 grammes (2 oz.).

Sig.—Daily dose: two tablespoonfuls of the solution; or, two dessertspoonfuls of the syrup, to be taken during meals.

The dose of mercury necessary is not hard to determine. We are not treating an adult, nor even a child, but a fœtus; consequently, small doses are sufficient, and experience has proved that this argument is accurate. What the *exact* dose for a fœtus may be is not yet known; but I can say that the cases in which I have succeeded have

taken half a centigramme (seven-hundredths of a grain) of proto-iodide, and even half that amount, daily.

This treatment should be kept up during the whole of pregnancy; on this point every one is agreed. It is only in points of detail that there are some variations. Thus, some authorities, such as Professor Pinard, advise an uninterrupted course of treatment without any rests. Others, among whom are Professor Budin and myself, prefer a treatment in periods separated by intervals of repose, such as twenty days of treatment and ten days of rest in each month. In this way the stomach has a chance to recuperate. Still others prefer an alternating treatment, giving first mercury then potassium iodide, with or without periods of rest.

You must not be surprised at these divergencies of opinion, or be deterred by them from following my advice. This question is not yet definitely elucidated; we are still in the stage of experiments and investigation. After all, these divergencies are only on questions of detail and of secondary importance. The preference for this or that preparation of mercury is insignificant so long as the mercury is given; and whether it is given continuously or in periods is also, in my opinion, not of first importance, as is shown by the fact that both methods have succeeded.

Lastly, let me say that you cannot be bound by *any* fixed rules, but have to adapt yourself to the peculiarities of each case. You generally have to do what you can, more than what you wish to do! Your favorite preparation may not suit your patient at all, so that you have to abandon it and give another one. Or you may prefer a continuous treatment, but find that your patient's stomach cannot stand it. So that, in this instance as in so many others in medicine, you have to be an opportunist and get along the best way the circumstances will allow.

# STUTTERING: ITS CAUSES AND TREATMENT.

CLINICAL LECTURE IN THE COURSE TO PHYSICIANS AND TEACHERS DELIVERED IN HIS POLYCLINIC, BERLIN, GERMANY.[1]

BY HERMANN GUTZMAN, M.D.,

Specialist in the Disturbances of Speech.

Gentlemen,—We take up to-day the subject of stuttering. It is one of the most important and practical subjects in the course, because of its comparative frequency, and, despite the persistency and seeming obstinacy to treatment in so many cases, the possibility of its being entirely cured, or at least greatly ameliorated, in the vast majority of cases, by methods properly adapted to the abnormal conditions that underlie the affection. It is a very large subject, and I cannot hope to convey anything like a complete idea of its entity, causes, and cure in a single lecture; yet I have preferred, as an introduction to the subject, to give a general presentation of the main features of the affection and of the physiological principles upon which its treatment must be conducted to be successful, and to take up special forms and complications of it afterwards. This will give you at once the basis upon which your detailed knowledge of the affection can later be built up, as we proceed with the practical lessons.

Our first patient is a little fellow of four years, who, as you see, keeps his mouth open as he breathes. This gives a less intelligent look to a face that is otherwise bright enough. When I have him open his mouth, you see the high palate. Our little patient came only yesterday, and so from a pædogogic stand-point I do not care just yet to make a digital examination of the back of the pharynx. We have, however, in the symptoms mentioned,—the expression of face, the mouth-breathing, and the high palate,—sufficient to justify us in the diagnosis of adenoid growths in the pharynx. Some such

[1] Reported by James J. Walsh, Ph.D., M.D.

anomaly of the throat or nose is not rare in stuttering cases, and while never the direct cause of the defective speech, it seems to be the point from which reflexly that heightened excitability of nerves which leads to the spasmodic conditions of stuttering has its origin. If I at once made a digital examination of these cases at their first visit, I would to a great extent lose my influence over them, because of the fear inspired. As the adenoid condition has existed for years, it can now afford to wait for a week or two. Its removal will not be immediately followed by disappearance of the stuttering, because, though it may originally have been an indirect cause, the spasmodic conditions reflexly induced have now acquired an independent character, and so will have to be treated for themselves. In the meantime the psychical suggestive element, so important in the treatment of stuttering, is not disturbed by loss of confidence in the medical man at the first visit because of painful or at least unpleasant associations. The training for the stuttering may go on, and the patient be in the best condition for the resumption of natural speech when the adenoids are removed.

When I have our little man tell us his name, he makes a movement or two with his hands, and then says "t'Herbert." His name is Herbert, but the vocalization of the "h" is difficult for him, so he uses the expedient of putting a consonant before it that comes easier for him to say. When I ask him to say "My mother," he says "M-m-my m-mother," making sympathetic movements at the same time with his head and hands. Note how superficially he breathes while talking. When I have him tell me the names of these pictures, you see this constant tendency to repeat initial consonants. We have here to do with the simplest form of stuttering,—this spasmodic repetition of letters. At his age it is extremely easy to cure it. He has not learned to regulate his breathing at the same time that he calls into play the effort of co-ordination required to talk, and so he must be taught.

Note the difference in his speech when I have him draw deep breaths and then after a moment have him talk during expiration. He came for the first time yesterday, so I shall have to teach him the method. I put his hand here on my chest while I take a long breath and have him note the expansion, then I put his other hand on his own chest and have him imitate me. He breathes entirely too superficially at first, but after awhile the breathing is deeper. Then I

catch him just at the beginning of expiration and have him say "My mother," and he does it without stuttering. I have him tell me his name in the same way, and am told plain "Herbert," without the "t." Now I permit him for a moment to resume his old habit of breathing, and the stuttering recurs.

We must, then, train and have him trained at home in deep breathing and talking during expiration. Here is an apparatus I sometimes use to tempt children to breathe deeply. It is an ordinary spirameter, into which I have them breathe. They soon learn that what, for the sake of interest, I call the elevator goes up much farther if they take long breaths, and I have them see how many breaths it takes to send it to the top. It is a play toy with a purpose, that interests the children and is often of great service in teaching deep breathing.

Our next patient is not a case of stuttering, but I present her in order to show you another difficulty of speech whose cause is very different from that of stuttering and whose correction requires very different methods. She is a girl of thirteen, from the country, stout and healthy looking, but without much intelligence in her face. When I ask her her age, she says, in a hesitating, slow, drawling tone, "Thirr-r-tee-een ye-arrs," mouthing the words so much that they are intelligible only to one who is accustomed to hear her talk. When she first began to come here, some two months ago, she was much worse than she is now. I show her some of these pictures, and you see how slow she is to tell me what they are. When she first came she called this a "toffee-tan," while she now says "coffee-can" rather intelligibly; the same for other words.

Here we have to do with what we call in German "stammeln," —*i.e.*, inability properly to co-ordinate all the motions necessary for the proper pronunciation of words, and a consequent defect in speech. Learners have a relative amount of "stammeln" in speaking a foreign language before they have become accustomed to the co-ordination necessary for the unusual sounds of a foreign tongue. Children have it because they have not yet learned the co-ordination required for proper pronunciation. This patient has simply, through defective intellectual development, remained a child beyond her years and has not yet learned to talk.

In such cases you will usually find a history of tardy development in other respects too. The teeth came late, the child did not learn

to walk until much older than is usually the case, and she is extremely backward in psychic matters generally. Here I have found by inquiry that our patient did not learn to walk until nearly five years old, and that at school she has practically made no progress because teachers could not give her individual attention and it was impossible for her to keep up with the other children. This, then, is "stammeln;" its differentiation from "stottern" is not difficult if but slight attention is given to the subject, but speech defects are sometimes all loosely thrown together under the rubric "stammeln" or "stottern" without further thought.[1]

To cure children of stammering,—*i.e.*, failure to pronounce properly,—time and patience are necessary. Normal children learn spontaneously in time. Children like our patient here require special teaching. Intelligence is insufficient to enable them to detect for themselves how others form certain letters and produce certain combinations of sounds; so these must be analyzed for them and put clearly before them. For instance, when our patient said "toffee-tan," she was shown that we did not in pronouncing these words put our tongue against the roof of our mouth just behind the teeth, as she did, but that the sound "k" was produced far back in the throat. After awhile she realized that for herself, and now has practised and can reproduce the sound. The same thing must be done for each of the defective sounds. This is a work of patience that only the most devoted teachers can accomplish, and usually proper advancement is secured only by the regular, thoroughly arranged work of an institution for such children.

To return to our stutterers. Here is a very different-looking patient from the last one,—a bright little fellow of twelve. In telling me his age he hesitates, opens his eyes somewhat, makes some visible efforts to talk and some movements of his head and hands, and finally, with a movement as of sitting down forcibly, tells me that he is twelve years old. When I have him stand up he goes through the

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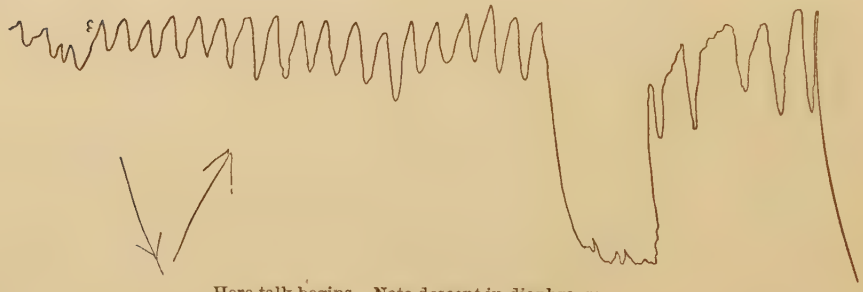
[1] NOTE BY DR. GUTZMAN.—The same confusion of terms, but to a much greater degree, exists in English stammering and stuttering, have even in the medical literature of the subject of defects of speech been referred irrelevantly to the condition we know as stottern, and for which stuttering would be the etymologically correct equivalent. An improvement in this matter has, however, been noticeable of late, and the English words are coming to be used with the meanings indicated above for the German words. It is in these senses that the translator of the clinic has always employed them.

same preliminary set of movements before answering, but replaces the final one, of straightening up on his chair, by coming down on his heels. These sympathetic movements are characteristic of stuttering; the voluntary impulse that is intended to lead to a certain movement, for the moment inhibited by spasm, flows over into other motor nerves and sets other movements into action. The final, almost spasmodic movement of a number of muscles seeking a point of resistance against some fixed object—the last impulse (stoss)—is a frequent accompaniment of certain forms of stuttering.

Note the difference when I have our patient sing. Here there is no difficulty. Note at the same time the difference between his breathing when he sings and when he attempts to talk. I have him recite exactly the same words as he sings, and the old difficulties return,—the gaspy, frequent, superficial breathing, the sympathetic movements, the appearance of intense effort at times followed by the quiet articulation of a few words, then the renewal of the effort.

Now let me demonstrate to you what is taking place in his thorax when he makes an effort to talk. I adjust a Marey drum, attached

FIG. 1.



Here talk begins. Note descent in diaphragm.

to a registering apparatus, so that it rests here just below the ensiform, holding it in position by these strips around his neck and waist. Here, because of the influence of movements of the diaphragm upon the abdominal contents and so upon its walls, we shall be able to catch and register the diaphragmatic movements. Now I have him simply breathe naturally, and you see the curve produced in the first part of the accompanying illustration (No. 1), and now I have him talk. Note what happens. Normally we talk during expiration, as may be seen on curve No. 2, showing the normal movements of the dia-

phragm. He begins at the end of inspiration, at least where normally on the curve expiration is just about to begin; but in his case the diaphragm, instead of going up for expiration, sinks lower and lower in a forced inspiration. Our patient, then, talks during the end of a forced inspiration, and then draws a hasty breath to begin once more, where normally expiration should begin but does not.

Fig. 2.

Normal diaphragm curve in normal breathing. Expiration as we talk normally.

This mechanism of the descent of the diaphragm, instead of its ascent, while many stutterers talk, has been demonstrated in Röntgen ray photographs, and may be plainly observed with the fluoroscope, so that it is not a mere mechanical delusion, caused by some anomalous spasm of the abdominal muscles, but an actual occurrence.

Our next case is a man twenty-two years old, who has been under treatment now for some two weeks. When he first came he was practically dumb in the presence of strangers or under the influence of the slightest excitement. When we ask his name, there is a moment of excitement, then the appearance of intense effort, and finally he is able to speak. I warn him of his breathing and then ask some other questions, and as he grows a little more accustomed to your presence you see that he answers much easier and the appearance of effort gradually vanishes. I have him read, and his pronunciation is comparatively smooth after the hesitant beginning.

I said the appearance of effort vanishes, because, as you may judge from the sweat on his forehead, an intense effort is yet required to overcome the tendency to spasm. He sweats easily and flushes easily, showing an irritative condition of the sympathetic nervous system. When he projects his tongue, it may be observed to be slightly tremulous. Not only is there a general tremor of the organ, but fibrillary tremors in the separate muscles of it. His soft palate is also in tremulous movement. His knee-jerks are lively and his cutaneous reflexes very active. There exists a generally heightened irritative condition of his nervous system, having its highest expression in the spasmodic incoördination that occurs when he attempts

to perform the rather complicated series of acts required for speech. In such cases the prognosis as to complete and lasting cure is none too good. The defect of speech is dependent upon the generally lowered inhibition of the motor nervous system, which is due to a congenital defect very probably, that cannot be entirely overcome. Such patients may be greatly improved, however, by teaching them to breathe properly, and much, you see, has already been accomplished in this case. He is eminently well satisfied with the result so far, and is greatly encouraged to continue the exercises.

I must warn you, however, not to put too much reliance on the early improvement in these cases. The nervous condition is one eminently liable to be influenced by suggestion, and usually is so influenced to a marked degree at the beginning of the treatment. But then comes a remission, it is often almost a relapse, and discouragement may take hold of both doctor and patient. In such cases, then, it is important to be prepared for a period of non-improvement after a most encouraging beginning.

Our next case is a variety of stuttering we have not yet seen; for, as in everything else, each patient who stutters must be the subject of special study for himself, and no general formula of symptoms will fit all cases. He is a little fellow of ten, whose general expression is not the most intelligent, who is, as you can see, a mouth-breather, and who has a high-vaulted palate. We have here doubtless, as in our first case to-day, adenoids. He tells me that he has gone to school regularly, but that he is only in Class 5 B. At his age he should be in Class 3 B. His stuttering has probably kept him back, as it is extremely liable to do, since his inability to express himself puts him at a decided disadvantage. I do not agree with those who think that stutterers are usually of lower mental calibre than normal. The characteristic speech of the mentally deficient is a drawl, not a stutter.

The dull, expressionless face of the mouth-breather in whom the presence of adenoids prevents nasal breathing is well known. As stuttering is sometimes a complication of the adenoid overgrowth, the two conditions will often occur together, but this must not be taken to signify that psychic deficiency is correlative to stuttering. Very seldom have I met phlegmatic people who stuttered, and in the few cases that have come under my notice the progress of the cases under treatment was most satisfactory; there were no remissions in improve-

ment when once begun, and the result obtained was lasting. On the other hand, stutterers are characteristically lively, nervous, excitable, and such people, even when they do not actually stutter, often heap their words together under the influence of excitement when they talk rapidly, producing a modified stuttering, from convulsive muscular overwork, as it were. One of the best proofs of this is the fact that in young, excitable, anxious patients stuttering is always at its worst; as they grow older and take things more as they come, without nervous anxiety, the stuttering becomes less noticeable, to recur with all its early energy at times in a choleric old age.

But to return to our patient. When he answers my question, you note, as before, the sympathetic movements and then the final "push," as he throws his head back and talks. There is an additional feature here, however; just before he talks there is a peculiar sound in his throat, a clucking or "slushing," as if the soft vocal cords had been pressed together and then forced suddenly apart. There is a somewhat explosive character to the sound, as if the air had suddenly found a vent after having been under pressure. There is here a sympathetic spasm of the laryngeal muscles, that forces the vocal cords together at the same time that the spasm of the diaphragm asserts itself. This closure of the glottis must, of course, be overcome before speech is possible; hence the sound heard just before he begins to talk.

When I have him whisper instead of vocalizing, you notice that the sound still occurs, and that there is a hesitancy in beginning a phrase, not so marked as when he talks out loud, yet distinctly noticeable. It has sometimes been said that stuttering does not occur during whispered speech, but, as you can see here, this is not absolutely true, and I have seen cases that were much worse than this. Stuttering during whispering usually occurs when there is a marked participation of laryngeal muscles in the spasmodic condition that ensues on attempts to speak. This preliminary spasm is, however, quite rare. It requires for treatment practically the same method as other forms of stuttering; practice in breathing will give the co-ordination so necessary for the complicated movements required for speech.

I have finally two extreme cases of stuttering to show you, young men of eighteen and twenty, who under the influence of the excitement of coming before the class will scarcely be able to utter a word.

In both cases you can see the supreme effort employed but in vain. In this case, to answer my question "How old are you?" the effort has brought out sweat on his forehead, he has gulped and gurgled, and finally has said, "Na—eighteen years old." To help himself out, he has used an auxiliary, or rather preliminary, syllable, that he has learned by experience to be the easiest for him to begin with. That even with this aid he does not achieve a great success the amount of effort required to answer so simple a question demonstrates.

This use of *fleck* words, as we call them in German, intercalary words I believe they are called in English, is a favorite expedient of stutterers. They have found that the position of all muscles necessary to pronounce a certain syllable is comparatively easy to them, and that, having made a commencement of vocalization, they can usually go on to the end of a phrase. They insert these syllables, then, and when they are short and insignificant the effect produced by their introduction is not striking. Many people who do not stutter, at least not vocally, but whose thoughts clog for a moment or to whom the conscious effort of continued speaking seems to require breaks, acquire the habit of introducing the word "now," for instance, in a way that is much more disagreeable.

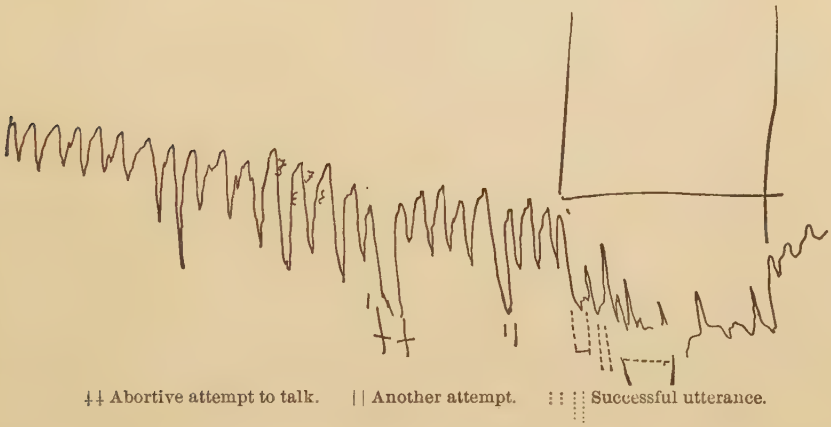
Sometimes, when these intercalary words have a definite significance, the effect produced by their introduction may be intensely comical. There is the story of the old general who intercalated incontinently the word "mama" into his orders, and especially when angry or excited. He is reported to have once said, when very angry, "The old fool mama thought that we mama would haul mama his chestnuts mama out of the fire mama." Kussmaul, who tells the story, called the symptom embolophrasie, and considered it the result of a lesion of the central nervous system.

Let us note in this patient, who is scarcely able to talk, so severe is the spasm that ensues on each attempt, what happens when he makes this effort. We use the Marey drum and a registering revolving cylinder, as before. Here you see (curve No. 3) the regular, rather superficial breathing at first, and then, when he is asked a question and attempts to talk, this marked descent of the diaphragm and its retention in this position so long as the effort continues. When he succeeds in uttering a word, this is not because his diaphragm ascends in a normal expiration. The utterance is effected by the use of the intercostal muscles and the accessory muscles of respiration,

these forcing some air out of the thorax. The severity of the condition here and the clearness with which the cause of it comes out show the necessity for the treatment by exercise in regular deep breathing.

I have still another case to show you but for a moment before we go on to the treatment proper. He is a lad of fifteen who developed his stuttering within the last few months and almost without warning. The condition is not severe. Such cases of stuttering about the

FIG. 3.



‡‡ Abortive attempt to talk. || Another attempt. ⋮⋮⋮ Successful utterance.

period of puberty, when the voice is changing, are not extremely rare, and the most interesting thing about them is their almost sudden beginning. An analogous cause would seem to be the occurrence of the second dentition, a period at which the first symptoms of stuttering are not seldom noted.

Now, you must have noticed that, while I have presented more than a half-dozen stuttering patients, not one of them is a female. Their absence has a very good reason; it is, that stuttering is much rarer in females than in males. Statistics of German school-children from various cities show that out of seven hundred stuttering children only two hundred, less than thirty per cent., were females. In adults the contrast is still more striking, and women constitute less than ten per cent. of the stutterers. The difference is evidently due to the fact that the breathing type, even in girls who do not yet wear stays, is more costal than costo-abdominal, and after the stay period is practically entirely costal, while that of males is originally and permanently costo-abdominal. We must bear this in mind, for it will be of use in the therapy of the affection.

And now as to treatment. Speaking requires a form of breathing somewhat different from that we ordinarily employ. It requires especially a lengthening of the expiration. Normally expiration and inspiration, as we can hear in the regular breathing of a sound sleeper, are about equal. For speaking inspiration must be short and quick and expiration prolonged. This quick inspiration cannot be accomplished through the nose. In speaking we breathe in and out through the mouth, so that the first thing is to have our patients open their mouths and keep them open during the breathing exercises.

Let us take our patients here and note the character of their expiration. We find it, in all of those who have not yet had any exercises with us, very much shorter than inspiration. Many of them, as soon as the full expansion of inspiration has occurred, let their thorax walls fall together, as it were, and in a few seconds expiration is past. If you try your own expiration, you will find that you can prolong it at least to fifteen or twenty seconds,—that is, most of you can, for I see some of you who have not done so. I wonder if those who now for the first time recognize how short their expiration is are not conscious of certain difficulties in talking fluently. Many people have such as the result of faulty expiration, though the condition never develops into anything so serious as stuttering.

I have had stutterers whose expirations were not longer than three seconds, and to whom the whole process of breathing had to be taught. In order to do anything with them they must be taught to expire slowly. The teaching is not a difficult task, as a rule, but one thing must be borne in mind. There are certain delicate people, usually of highly organized nervous systems, who when they attempt consciously to control their breathing for a time become faint and giddy. In such cases the breathing exercises must be taken up very gradually. It may also happen in nervous people that slow expiration is followed by severe palpitation of the heart. Especially is this brought about in such cases by holding the breath for any length of time. There must, then, be no long holding of the breath in such cases, and the occurrence of palpitation of the heart must be the signal to stop the exercise for the day.

If we wish effectively and easily to teach the process of breathing, it must be carried out under the care of the consciousness; for this costal breathing is necessary so that patients may feel the rise and fall of the thorax walls. You must call patients' attention to the

rise and fall of the ribs in this type of breathing, and especially they must learn to control the fall of the ribs, so as to have a constant supply of air at command. Even little children may be taught costal breathing by having them put their hands on your thorax, note the rise and fall that occurs in its walls, and then be asked to imitate it.

Costal breathing may be exaggerated and more air for expiration secured by certain movements of the arms. If I lift the hands straight out from the body, palms down, and continue the movement until the *backs* of the hands touch above the head, I increase the circumference of the upper part of the thorax by some centimetres. This increases the vital capacity of the thorax and gives a larger reservoir for air. Remember that, if only the palms of the hands are brought together above the head, there is a compensating muscular mechanism that acts on the scapula and the movement is accomplished without enlargement of the thorax. This exercise with the hands in connection with the breathing is very useful.

I now put our patients through these exercises and call your attention to defects. Here is one who has never taken the exercises before, an extreme stutterer. Note how he allows his thoracic walls to fall together as soon as he begins expiration, and then attempts to continue expiration by forced use of the accessory muscles of respiration.

Note the sound that is made on inspiration in this case. It indicates that the vocal cords are in the whispering position. This must not be allowed, for there is a certain effort required for it, and it predisposes to spasmodic contracture at the beginning of expiration if any muscular co-ordination should be attempted, as in talking. The glottis must be widely opened and the mouth freely open.

Here is a little patient who thinks he is attaining the acme of costal breathing by lifting up his shoulders. I need not tell you that this does not increase vital capacity, and that the muscular effort required cannot but interfere with the nice co-ordination of muscular effort required for speech if it should be attempted under any such conditions.

These simple exercises in breathing, taking care especially that expiration is prolonged until a certain amount of familiarity with the control of muscles necessary for prolonged respiration is acquired, need not take more than a few minutes the first day with older patients, though these simple exercises must be repeated each day, not

only with you to control them, but also by themselves or under the control of friends. You may begin at once on some vocal exercises, so as to give patients the confidence necessary to encourage them to continue their efforts faithfully; for it does not take long to demonstrate to a patient that the method can be of great help to him, and this suggestive element has an important therapeutic effect.

We first have the patient break inspiration up into several divisions, breathing in in successive whiffs, to exercise the respiratory muscles in stopping and starting; then we have him expire in the same way, in a number of puffs, being careful, however, of one thing: the breath must not be held in by closure of the glottis, but must be retained by the equilibrium of pressure between internal and external air that ensues when the muscles hold the thorax in a certain position. This is very important and must be insisted on, for the spasmodic closure of the glottis to retain the air is one of the false muscular manipulations that already make speaking so hard for the stutterers.

Now comes the teaching of co-ordination in the use of the muscles necessary for vocalization. Some have thought that singing would do this; but, as I have shown, many patients do not stutter when they sing, so that exercise cannot avail much. Others make speaking easy for stutterers by introducing a certain rhythm into speech, and there is no doubt that this accomplishes a good deal. Most stutterers do not stutter while the rhythm is on. As Kussmaul has pointed out, the rhythmus acts as an efficient will regulator. When the rhythm cannot be any longer employed, however, as is the case in ordinary conversation, the old faults return.

I have thought it much more rational, since there is question of teaching co-ordinate movements, to analyze these and exercise the various phases of them separately. Every attempt at co-ordination is the signal in the stutterer for a spasmodic contraction, but, if we exercise the muscles that must afterwards be co-ordinated at first separately, we avoid the spasm. When we expire with the production of only a slight whispering murmur,—*i.e.*, expire audibly,—we use only the musculi vocales or thyreo-arytænoidæi; when we whisper loudly the crico-arytænoidei laterales come into action; when, finally, we vocalize, "talk out loud," there remain only the musculi arytænoidei to come into play.

This, then, is the course of the exercises. First, attention to pro-

longed expiration; next, audible breathing; then, whispering; and, finally, vocalization, always of simple sounds and *always* during expiration. With that system I have found that any stutterer could be greatly benefited and most of them practically cured. There are details in its employment that I cannot go into to-day. Especially may the demonstration to adults, either by external palpation of the larynx or, as I have done it in certain cases in more intelligent patients, by autolaryngoscopy of the spasmodic condition of their muscles during attempted phonation, in order, by educating their muscular sense to a realization of the sensations of this faulty position, to enable them to correct it.

And now at the end comes the objection that will occur to many people's minds,—that this is only a system of muscular exercises which is supposed to compensate for a nervous defect; for stuttering is undoubtedly due to certain nervous failure to control muscular action. In recent times we have learned how much in other nervous defects—for instance, the incoördination of tabes—may be compensated for by muscular training. This principle my father had applied to the treatment of stuttering long before the movement therapy of tabes or analogous modes of treatment were thought of.

It is well to recall here what the distinguished physiologist Du Bois-Reymond said of training, "By training we understand usually the frequent repetition of a more or less complicated movement of the body under the direction of the mind, or even a training of some action of the mind alone, so that it may be more easily done." He then insists that bodily exercises are not alone muscular gymnastics, but also nervous gymnastics. "We may accept," he says further, "that the normal muscle, as a rule, promptly obeys the nerve, and that its state of contraction is predetermined at any given moment by a preceding excitation of the nerve. As the nerves bring only the impulses that originate in the motor ganglia cells, it is clear that the true mechanism of the co-ordinate movements has a place in the central nervous system, and that training in such movements is nothing more nor less than training of the central nervous system."

We may feel perfect confidence, then, in our method, since it is founded on principles that one of the greatest of physiologists approves, and take it up with the feeling that a misplaced enthusiasm is not leading us to idle efforts to overcome an irreparable natural defect.

# PHYSIOLOGY OF CIRCULATORY DISEASES, WITH REMARKS ON TREATMENT BY DIGITALIN.

BY HENRY BEATES, M.D.,
Of Philadelphia.

In directing your attention to the subject which I have the honor of considering with you this evening, the liberty is taken of briefly reviewing the minute structure or histology of the principal elements involved, and of advancing, from a *physiological* stand-point, certain factors, which, if known, have not, it seems, been accorded the importance they demand in the vast domain of *circulatory* function, and, as these characteristics belong chiefly to that side of the circulatory system intimately associated with the propulsion of the blood mass, the heart will first command notice. Its musculature, being of the involuntary type, presents cellular differences of pronounced character, when contrasted with voluntary-muscle fibre. The cells are mononuclear, the nucleus occupying a central relationship, while the ends are fibrillated or serrated, and connected with juxtaposed cells by a fibril of one merging into a corresponding process of another. Each cell is enveloped with a delicate membranous structure, which is not, however, a separable sarcolemma, as is found in voluntary muscular tissue, and between the cells, like a scaffolding, useful for support, are processes of connective tissue on which are located capillary lymphatic and blood radicals. This connective-tissue element is a continuation, so to speak, of both the pericardium and the endocardium, and this relationship explains how, by continuity of structure, inflammatory and degenerative processes primarily originating either without or within the heart may ultimately involve the entire structure. Please observe the peculiarity of the *relationship* of the blood and lymph radicals to cardiac-muscle cells; they are situated *between* the cells, and do not, as in striated voluntary muscle, supply pabulum more directly to the element itself.

Cardiac-muscle cells, therefore, derive nutriment from the fluid surrounding them, and appropriate it by means of imbibition and inherent vital power, in a manner similar to that of the cells constituting cornea and cartilage. The importance of this is seen when the peculiarity of the ultimate distribution of the coronary arteries is recalled, and which consists in the fact that the terminals of this vitally important source of supply of pabulum do not anastomose. It is apparent, therefore, that any interference with the free circulation of pabulum in any given area would, because of the impossibility of establishing a collateral circulation, result in the deprivation of nutriment and consequent *local* degenerative changes. The corollary naturally insinuates itself here, that, as the nutriment essential to the normal cardiac functions is supplied by the coronary channels, it is the normality of these vessels and their function upon which depends that of the heart.

Physiological investigation demonstrates that sudden obliteration of one coronary artery is followed by marked irregularity of ventricular contraction; sometimes there is temporary total arrest, but, more commonly, *localized* imperfect but frequent efforts at contraction. This disturbance, noticeable in limited areas, is additional proof of the peculiarity of relationship existing between the cardiac cells and their channels of food-supply, and also of the terminal nature of ultimate distribution. The completeness of each cardiac contraction depends upon the perfect fulfilment of coronary function. This is proved by obliterating a coronary artery, which, when done, is followed by a progressively diminishing intraventricular pressure during systole, and a proportionate increase during diastole; and, when these conditions obtain, there is irregularity of contraction both as to time and force. Observe that these phenomena apply largely to the force of the contraction and that, while the rate is also influenced, other factors are operative; but the chief physiological fact to be demonstrated is, that the *force* of the heart-beat is directly proportional to the *normal volume of blood flowing through the coronary system with each cardiac cycle;* therefore, the nutrition of the heart, depending upon the food-supply, is, in turn, vitally related to the normality of the coronary vessels. These arteries are so arranged anatomically as to depend upon *arterial tension* for their complete supply of blood; therefore, when, after ventricular systole, the aortic valves close and the recoil of the aortic

column occurs, if any condition exists that results in diminution of the force of this recoil, or, what is the equivalent, the normal supply of the necessary volume of blood to the cardiac viscus, there *must* follow, in the widest sense, a proportional diminution of cardiac function. Whether this results from disease *at* the coronary orifices, along their channels, or anywhere in the peripheral arterial area, makes no difference. *It is diminished arterial tension, primarily existent*, that underlies cardiac degeneration in almost every instance. True, we do encounter primary cardiac disease; but it is the exception, not the rule, and occurs under conditions radically different from those under consideration.

Let us now review some striking features of the arterial system. We here must cognize the "three-coat structure,"—*i.e.*, the intima, with its peculiar smooth internal surface composed of longitudinally arranged endothelium, resting upon an outer connective-tissue structure, conspicuous from the fact that its quantity varies with the size of the artery, and must, therefore, possess some relationship to the physical function of that artery; also because it is intimately related to an elastic lamina, which latter becomes the base upon which the endothelium rests in arteries of smaller size, for in these the connective-tissue structure is no longer needed and is, therefore, absent; so that, where a certain physical property is served in arteries of larger calibre, there is a protective or supplementary histological factor operative by the presence or absence of a connective-tissue layer, as this is functionally related to the elastic lamina. In the aortic intima this elastic lamina is very conspicuous by its absence, just as the connective-tissue layer is by its presence, and thus is beautifully demonstrated the fact that arteries possess a very decided and positive function, a function to which almost no attention, much less importance, has been accorded. Reference is had to the power of *propulsion*. This peculiarity of minute structure also presents conditions connected with nutrition that afford physical states possessing tendencies to *local* alterations of cell life, which mean much in correctly interpreting trophic diseases of the circulatory system. This feature is well illustrated by the commonly met degenerative changes existing in the aorta, and which are in sharp contrast with lesions of other areas of the circulatory system. One point regarding the elastic lamina: the smaller the artery, and the less we have of connective tissue, the more pronounced is the corrugation of the elastic

element,—*i.e.*, the more that calibration of an artery becomes essential, the more perfectly is the physical condition for the fulfilment of this function developed.

The media is the largest of the three coats, and possesses a muscular system that is proportionately developed to the elastic property of an artery, and thus, the arteries becoming smaller towards the periphery and a propelling power being required, we find the musculature better and better developed. What does this mean? Unquestionably, that, after leaving the aorta, the onward movement of the blood mass is directly dependent upon the inherent power or ability of the arterial channels to propel the blood to the ultimate cells of every organ of the economy.

If this be true, the importance of the heart as the organ for propelling the blood must be less than is commonly believed, and just as much less as there is need of increase of propelling power in the ultimate ramifications of the arteries. If the heart by a single contraction shot, so to speak, a few ounces of fluid into a blood column, the onward movement of which is retarded, opposed, and prevented by a rapidly increasing friction, greatly augmented by the numerous subdivisions, and which onward movement depended upon the momentum given to this mass by the force imparted to these few ounces by a contraction of the left ventricle, the physical construction of the heart and aorta would be utterly inadequate to withstand the strain, and indeed could not possibly accomplish such a result and not suffer immediate laceration or rupture. The proportional presence of muscularity to given arterial function, then, demonstrates a vital property of the arteries which must be accorded, if a comprehension of the physiology, pathology, and treatment of diseases directly and indirectly related to this vast system is to be attained.

This arterial muscular system is of the involuntary type, and possesses a perfect nervous and vascular supply as complete as does the heart itself, and, like this latter, these vessels contract and expand as the especial areas, which they supply, demand. Think of the multiform hydrostatic variations which every change of position and every movement require. Does the heart, under these ever-varying requirements, give evidences of compensatory potential deviations? And yet these compensatory—or, rather, complemental—vital phenomena must on the instant be supplied, and, if the heart does not accomplish it, what does? Manifestly, *the arteries*. Now recall how

the nutrition and achievement of cardiac function—in a word, its life—are directly dependent upon constancy of arterial tension; not arterial pressure, but arterial *tension*. Arterial pressure is an expression of the *weight* of the column of blood as indexed in the aorta, while arterial tension is the expression of the propelling power of the arteries. Arterial pressure indicates a column of fluid in a relatively retarded rate of progress; arterial tension, of normal rate of progress and the constancy of related potential energy, and thus is the *error* of the teaching and belief that in the condition of rigid arteries there is increased arterial tension demonstrated and explained.

In this condition, with arteries comparatively empty and veins preternaturally full, there is *never* increased arterial tension, but, on the contrary, very markedly *diminished* tension. Apoplexies, *venous* apoplexies, under these conditions, are, therefore, not provoked by augmenting arterial tension, but most certainly, other things being equal, prevented.

The adventitia, a white connective tissue of fibrous type, is the coat containing the vasa vasorum, or vessels of food-supply, essential to the vitality of the artery. The nerve-fibres traverse this coat and extend into the muscular layer or media, and thus there is completed, what should be considered to be, an organ possessing a vital function, not of a minor but most important character. Nutrition in general, normal blood-supply, completeness of functional achievement,—in a word, life and health,—depend upon normality of arterial function, and not, in the sense here advocated, of the heart.

Why in those rare instances of purely fatty cardiac metamorphosis does the circulation continue almost normally until that time when, in apparently good health, the next cardiac pulsation does not take place, and the victim falls a corpse before he reaches the ground? There have been no dropsies, dyspnœa, or markedly distended veins, and yet on autopsy the cardiac musculature evinces such a degree of advanced retrograde change that a normal cell is with difficulty found. The organ is flabby, and frequently, under the microscope, discloses fragmentation and segmentation. If the arterial system be studied under these conditions, the beautiful mechanism briefly outlined above will be found, by reason of compensatory development, abundantly ample to answer the question. What maintained the circulation while that which is too generally believed to be the pro-

pelling organ of the blood was slowly and progressively becoming a structure that to maintain such a function was a vital and physical impossibility? Manifestly, *the arteries.*

Without further occupying time with details, let us apply the principle of circulatory function thus very briefly set forth, and inquire whether exceptions obtain in explaining pathological conditions.

The primarily fatty heart, with its absence of symptoms, has been cited, but contrast with this valvular diseases and the characteristic coexisting dropsies. Why in the former, when there is scarcely any longer a blood-pump, are there no dropsies, while in the latter these constitute a very conspicuous feature? also, why the absence of respiratory distress in the former, and its very persistent presence in the latter? The answer is in the fact that, circulatory equilibrium being maintained, neither dropsy nor dyspnœa is possible. This being so, why is not circulatory equilibrium maintained with a leaky but fairly potent heart, when it certainly is with a pump whose structure is gradually becoming a foreign or inert mass? The *arteries maintain* the circulation, and, by virtue of their ability to respond to a reflex, they calibrate and propel and thus circulate the blood.

The feeble tap of a degenerating heart, because the pressure of fluid is equal in all directions, enables an arterial system to respond reflexly to the requirements of the economy and convert kinetic into potential energy. Other physiological factors are doubtless operative, but in the main this obtains.

In valvular diseases the concussion or impulse that calls into play the arterial reflex is *constantly minus;* therefore the nutrition of the arterial system first diminishes, because of the consequences of continual subtraction from vasa vasorum function; then, secondary to this, for reasons now evident, the heart suffers, and we have, affecting the mass of fluid, a diminished reflex-exciting impulse, supplemented by an already degenerated propelling organ, the arteries, and thus is the arterial side necessarily comparatively empty, its normal tension minus, and the venous side distended; therefore dropsy, local, and ultimately general, associated with dyspnœa, passive hyperæmia, and its consequential distress and ultimate loss of life must supervene.

Pathological study of this whole group of cases discovers condi-

tions positively proving the correctness of the proposition here advanced. Let me repeat, that balance which in health exists between the venous and arterial sides of the circulatory system, and upon which *all* function so largely depends, is what is known as *circulatory equilibrium*. Disturbance of this must in time be followed both by alteration of structure and interference with function. This latter usually, other things being equal, first manifests itself, while the former occurs as an epiphenomenon and is a consequent. At a glance it is thus evident that during middle life many diseases regarded as largely functional are prone to present: also, that these centre around disturbed function, either special or more widely involved, and manifest themselves by diminution of the normal standard of achievement. For obvious reasons, those functions which are an expression of the highest type of metabolism are the earlier to fail and, in the writer's experience, are first encountered in connection with the cerebrum. The beginning of this form of trouble is apparent in impairment of ability, and is recognizable on the part of the patient, who for the first time realizes that only with difficulty is he capable of accomplishing work requiring executive power which formerly was achieved with pleasure and ease. The cerebrum is too readily fatigued, and memory is correspondingly lessened. Vision about this time requires the aid of lenses, because the normal plasticity of the accommodative apparatus is slightly below the normal and incapable of complementary action. In more advanced instances the superficial veins give evidence of preternatural fulness, and not infrequently the urine contains albumen and even hyaline and epithelial casts. Now there is decided lowering of vitality, and ambition and spontaneity give place to relative lack of interest and lethargy. This you may say is only premature old age, but when it is remembered that these symptoms are an expression of circulatory deficiency, and advanced only under such a condition, you will agree that, while senile in type, they are but an expression of diminished function directly resulting from a want of normal supply of nutriment. This point is perhaps the better made clear by quoting from another article written some time since in connection with a different phase of the subject. The term "senile heart" refers only to that retrograde change of the myocardium which is consequent upon those diminished metabolic processes invariably attendant upon age,—that is, the normal lessening of inherent cell

activity which takes place because the *life unit* has reached its "threescore years and ten."

The totality of phenomena indicative of such, by reason of the complexity of relationship characterizing the organology of the human economy, fortunately represents a process which is operative under two widely different conditions, and may, therefore, be symptomatic of a general or systemic age and a cardiac or limited senility constituting a *special* but prematurely old condition; and so in this instance, occurring in middle life, other things being equal, the apparently senile symptomatology is expressive not of old age, but of impairment of function. If this were expotential of aged cells, nothing by treatment could be achieved, because, if pabulum were supplied, the cells, having lived their life unit, would be incapable of appropriating the essentia vitæ. Cells, however, deprived of an adequate supply of nutriment will in time degenerate and die, and, according to their importance in the economy, life itself is impaired or destroyed. The question now naturally offers, what determines these apparently senile phenomena at a time when the *vital unit* is still in its prime? In a general application of the law of cause and effect, the answer is embodied in the phrase "circumstance and environment," and this results in a disturbance of the circulatory equilibrium, and is associated with functional failure—partial, it is true—of that property, *calibration*. The movement of the arterial wall necessary to accomplish this, like that of the cardiac wall, is essential to the proper circulation of both the lymph- and blood-channels, and is comparable, indeed, with the parallel motion of joint-function. It is scarcely needful to remind you of the so-called ankylosis, so constant an attendant upon an unused joint, and yet no less important is it to establish arterial movement, so to speak, in order to secure perfect freedom of action, than the same under conditions of the joints. Adverting to the cerebrum, we have forgetfulness in middle life, becoming, later, when acquired local senility finally supervenes, as grave as that which is the expression of true degeneration of the cortex, the only difference clinically being that the former disappears under treatment while the latter does not. What has been stated of the cerebrum is equally applicable to any organ or group of organs, and it is not necessary, therefore, to engage time further with superfluous specializations. The principle is the same.

The point of vital import is, that, if such degenerative tendencies are functionally established and not corrected, true organic retrogression will certainly ultimately follow as a direct consequence.

How can this be successfully met? Remember this whole group of diseases is that which is expressive of disturbance or partial destruction of *circulatory equilibrium.* Only by restoring arterial function,—this is the principle.

It is evident, then, that at this stage of incipient or functional circulatory disease, whether it affect an organ or a system, we have confronting us what is in reality a prophylactic treatment. This term is used in the sense that when nutritional circulatory disease presents, *before the vital unit is so far expended as to permit of organic or retrograde change,* we can, by re-establishing circulatory equilibrium, restore metabolism to the normal, and thus prevent premature and permanent diminution of function. The same principle of treatment is applicable, of course, in those circulatory diseases which are the result of special primary organic disease, such as valvular lesions and the secondary cardiac troubles, aneurism, arterial infiltrations and degenerations, etc. Under these conditions some dyscrasia or infection immediately establishes physical underlying causes, which become constantly operative as factors preventing the supply necessary to replenish wear and tear. Mitral or aortic lesions consequent upon an acute inflammatory rheumatism or a scarlatina serve as more common examples. What is the real disturbance when such physical changes exist? Practically the same as when the coronary artery is ligated, because a diminution of the *force* of contraction of the heart results in a correspondingly reduced intensity of impulse, which calls forth the arterial reflex; this occasions a subtraction from the force of the arterial column, and prevents the coronary terminals from being fully supplied; all of these culminate in an enfeebled cardiac pulsation, and, as the various tissue elements, being thus deprived of pabulum, sooner or later lose their vigor, we have *intraventricular pressure becoming less during systole and greater with diastole.* The venous system gives evidences of this by distention. The superficial veins are full and tortuous, passive hyperæmia is now established, and health and strength are rapidly undermined. From this stage on, nutritional disturbances assume an organic type, and degenerations occur which *prematurely* terminate life. The full force of this retrograde tendency is sustained,

for reasons now plain, by the arterial system, so that, by the time apparently slight functional diseases manifest themselves, there are already established nutritional or trophic lesions everywhere throughout the circulatory circuit.

For these lesions, arteriosclerosis, endarteritis obliterans, atheroma, and even aneurism, to say nothing of the miliary form so common in the cerebral structure, we are taught to avoid the exhibition of circulatory stimulants, because of the alleged existence of increased arterial tension.

Sufficient has been said indirectly regarding this prevalent but erroneous belief. When the propelling function of the arteries is less than normal, there cannot possibly be an *augmented arterial tension*, but, on the contrary, there is diminished tension. The contained blood is not flowing onward into the venous side in a manner that can only be *normally* accomplished when the ability of a given artery to perform its function to the full is not prevented, and thus eventuates marked lessening of the standard quantity of blood in the arterial channels, and a correspondingly increased volume in the venous side. This means, therefore, the existence of conditions the most favorable to the rapid and progressive development of nutritional circulatory diseases, together with their direct degenerative complications and serious indirect consequences.

Briefly let us consider the treatment. Of the whole field presented, correction of unfavorable circumstances and environment having been secured, the application of the principle is required. This consists in restoring to the circulation its lost equilibrium. Is this accomplished by the administration of remedies known as cardiac stimulants? No, for these contain many active principles antagonistic the one to the other, and must, therefore, as they *by chance* happen to contain the essential principle, either benefit or aggravate the difficulty. Clinical investigation has enabled the writer to prove that in digitalis there exists a derivative, unfortunately known as digitalin, which *does* restore to the circulation *equilibrium*. The tests proving this were applied in those instances where digitalis, the crude drug, and its still more crude official preparations, exhibited by competent clinicians, utterly failed, as did, also, strophanthus, adonis vernalis, convallaria, cactus, caffeine, nitro-glycerin,—in a word, the cardiac stimulants,—to achieve the desired end. As digitalin is a name applied to many different principles separated from

crude digitalis, it is necessary to remark that that which accomplishes the results here indicated is known as *Digitaline German* (Merck). It is prepared by precipitating from an infusion a tannate, which subsequently is subjected to a complex process which it would be foreign to the purposes of this contribution to detail.

This derivative, administered in doses ranging from one-tenth to one-half grain, from four to six times daily as conditions require, accomplishes results that no other plan of drug treatment can even approach. Particularly is its usefulness demonstrated in that range of cases in which we are taught that digitalis, the *crude drug*, should never be administered, because of the *falsely alleged great arterial tension*. The writer has held in abeyance for years, by maintaining circulatory equilibrium, slight apoplexies, which, prior to this being achieved, had repeatedly occurred, and not only this, but the whole system *becomes normally related to the vital unit* and an all-round degree of health is restored, that converts an invalid into a comfortable and relatively useful being.

# THE TREATMENT OF TUBERCULOSIS.

CLINICAL LECTURE DELIVERED AT THE CHILDREN'S HOSPITAL

BY PROFESSOR J. GRANCHER, M.D.,
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## LECTURE V.

Gentlemen,—You will have seen, from what I have said concerning the substances that are supposed to stimulate the nutrition of the body and thereby lead to the sclerous transformation of tubercles, that, after all, we are obliged finally to fall back on food, and base on its action our treatment of tuberculosis.

What food must we choose for our tubercular patients? Must we choose for them, or is it preferable to let them follow their own tastes and instincts, allowing them to eat more or less of what they wish, so long as it is food?

There are, no doubt, many consumptives who can be trusted to select their own regimen, but their number is evidently a limited one, and is composed of patients who have learned how to take care of themselves, to watch their digestion carefully, and to treat each slight disorder of the stomach or bowels. How to digest properly, and to obtain from the digestive organs a maximum result with a minimum of effort, is a matter that does not come by intuition; the physician must teach his patient how this can be done, or the latter will quite unconsciously make the most disastrous mistakes. Each patient is apt to have his own pet theory on the question and to carry it to excess: one will think that wine gives strength and will drink it in large quantities, while another will have equal faith in milk or bouillon, which he will consume *ad libitum*, although such a regimen may not be at all the one that will suit his gastric condition, a factor that varies with each patient.

They must, therefore, all be started on the right path; and, for this purpose, let me remind you of how food has been classified as regards facility of digestion and nutritive value.

A good article of food is one that can be easily and promptly digested and that has a high nutritive value. Let us look in the tables of Leube and Penzold for those that fill these conditions. Facility of digestion is indicated by the length of time food remains in the stomach; this period is two hours for pure or alkaline water, weak tea, coffee, cocoa, and milk,—all liquids, and taken in moderate quantities (from one hundred to two hundred grammes or twenty-five to fifty drachms); this is also true of Leube-Rosenthal's dissolved meat, peptones dissolved in water, and eggs.

To these articles I, for my patients, add the pulp of raw meat, which for nutritive value I consider preferable to all other forms of meat. In the tables mentioned above, hashed raw meat only comes in the third rank, and requires three or four hours to digest; but this is hashed raw meat, not pulp, and the two things are quite different.

Let me tell you in detail how raw meat pulp should be prepared. The process requires care, and should be seen to by the patient himself or by some one who can be fully trusted. When it is prepared, the patient can take it in any way he likes: in balls rolled in sugar, in cold or warm bouillon, spread on bread (with or without jam), mixed with brandy or the yolk of an egg sugared and flavored with punch, or, best of all, in a warm thick soup (potatoes, peas, lentils, beans, etc.).

There are three steps in the preparation of raw meat pulp.

1. The piece of beef should be scraped with a blunt knife, the fibrous tissue being removed as fast as it comes to the surface.

2. These scrapings should be mashed in a mortar of stone, glass, or marble.

3. The meat should then be spread over a sieve and squeezed gently through with a spoon or ladle.

The result of these manipulations is a meat pulp without shreds or lumps, the perfection of an article for digestion and nutrition. In cold weather the day's supply can be prepared in the morning, if it is kept in a cool place; still, as it spoils very readily, the preferable plan, even in winter, is to prepare it for each meal. This is absolutely necessary in summer.

Through fear of tapeworms a similar pulp is sometimes made from mutton, but the article is not so satisfactory. I must say that I do not share the apprehension shown by many patients and physicians concerning tapeworms. Even if one were conveyed by beef

pulp, it is a very simple matter to get rid of it, and such an accident need worry no one. On the contrary, many patients would derive benefit from the extraordinary appetite that often accompanies the presence of a tapeworm. I know of three cases in which a tapeworm appeared to do much more good than harm, and where the stimulation to appetite caused by the parasite was distinctly advantageous.

Beef pulp, with eggs, form the basis of what we have called the "recovery" allowance of food for a consumptive. Taken in quantities varying from one to two hundred grammes or twenty-five to fifty drachms it does not overload the stomach, can be combined with the regular meals or eaten at other times, can be readily digested and well assimilated. I consider it infinitely preferable to meat powders, of which the patient quickly grows tired, whereas meat pulp will often be taken with an effort at first, but will be more and more readily taken in increasing doses as time goes on; it is also preferable to peptones, that are apt to be impure, to the Leube-Rosenthal solution, and to beef tea,—all of them less natural and less easily assimilated preparations. Care must be taken, especially at first, to interrupt its use for a few days if there is diarrhœa or marked distaste for the preparation, or to lessen its dose, or when resuming its use to do so in small (teaspoonful) doses which can be gradually increased. It should always be not only fresh but pure; the person's hands and all articles used in its preparation should be perfectly clean. I have seen several cases of acute indigestion and intestinal intoxication, with vomiting, jaundice, liver congestion, and diarrhœa, which I could only attribute to unclean preparation or to fermentation of the beef pulp.

As to beef juice prepared by pressing small pieces of beef that has been lightly scalded over a bright fire, I use it only to take the place of beef pulp for a few days each month in some cases, in order to give the stomach a rest and avoid dislike of the pulp. It is a preparation consisting mostly of water, a little albumen, salts, and coloring matter of the blood, and is far inferior to beef pulp, eggs, and even milk.

Blood powder and even fresh blood taken at slaughter-houses do not inspire me with much confidence, and I do not use them in my practice.

But, on the other hand, fish, and particularly fish that is not oily and has delicate tissues, such as the sole, whiting, or pike, is an ex-

cellent nitrogenous article of food that is readily digested except by some stomachs that will not tolerate fish at all.

Eggs are as valuable as raw meat, since they contain a great deal of albumen, and fat in a finely divided and easily absorbed condition; and, since it often happens that a consumptive's stomach has trouble in handling animal fat and even butter, the yolks of eggs, combined or not with cream, are a very precious resource to fall back on. I advise at the first meal in the morning two or three soft-boiled eggs (almost raw) beaten up in a glass; this is readily digested, and can be combined with coffee and biscuit or toast. This sort of breakfast is preferable, in digestive facility and nutritive value, to soup or chocolate, which are a load to the stomach and of inferior nutritive value. It is advisable not to fatigue the stomach at the first meal in the day, as it should be empty and rested for the mid-day meal, which is the most important of all. Yet the early breakfast is also important; it should be, as I have said, small in bulk. A little meat pulp, or cold meat, can be added to what I gave above, or the coffee can be replaced by a cup of bouillon.

Milk in moderate quantities (from one hundred and fifty to two hundred grammes or thirty-seven and a half to fifty drachms) is also a good article of food, though much inferior to the two of which I have just spoken, as it contains far less nitrogen per volume. I look on it rather as an accessory to the treatment, as a vehicle for other things,—meat, eggs, farinaceous food, etc.,—and I advise its use as a drink at meals, provided it is taken slowly, in small mouthfuls, and not more than from two hundred to two hundred and fifty grammes or fifty to sixty-two and a half drachms at a time. It is also useful when we desire to give the stomach a rest for a few days on a partial diet. I think it a mistake at any period of the disease to make milk the basis of treatment. Milk, which is perfectly adapted as food for an infant, as it suits its digestive power and contains everything that nutrition requires at that time of life, is far from being fit for the "working" allowance of a grown person, and, *a fortiori*, for the "recovery" allowance of a consumptive. I even think it a mistake to allow such patients to take it when they like between meals; no real benefit can be obtained from this practice, which keeps the stomach constantly at work without the intervals of rest that are indispensable.

Asses' milk and goats' milk, of which so much has been said, are

also only auxiliaries; the former, lighter, sweeter, and containing less fat, is particularly fit for use when a stomach is momentarily overworked; the latter is rather a fatty form of food, but its smell and taste are repugnant to many patients, and I much prefer to give fat in the form of eggs or cereals. As for milk powders, humanized milk, and peptonized milk, I should advise them only in very exceptional cases, and even less Ebstein's artificial milk. Koumys is sometimes of use when appetite is lacking and the intestine is susceptible and readily loosened. Kephir has appeared to me useful as a remedy when there is hypochlorhydria. It is needless to say that when milk is used none of the ordinary precautions of sterilization should be omitted; these precautions will not change its digestibility at all.

The various cereals are an invaluable complement to the diet of a tubercular patient; without their help and that of green vegetables we should have trouble in avoiding satiety, since, although the quality of food is important, a variety is none the less so, and, although I know of patients who have followed a uniform diet for years, they are exceptions. We must not forget that we have to deal with a class of patients who are all dyspeptic.

All the cereals contain (in varying proportions) nitrogen, fat, and hydrocarbons, the latter in abundance,—from 50 to 80 per cent. Bread contains 6 or 7 per cent. of albumen and 55 per cent. of hydrates of carbon. Barley is richer still—11 per cent. of albumen, 64 per cent. of hydrocarbons. The composition of rice is, however, of special interest to us in treating tuberculosis. It contains 7 per cent. of albumen, 0.9 per cent. of fat, and 77.4 per cent. of hydrocarbons; of all the cereals it contains the least fat and the most hydrocarbons, and, in addition to this, its grains keep their form by a very delicate framework that is completely softened by being cooked in water, so that rice leaves no residuum after digestion and never fatigues the stomach. Think how many races live on it almost exclusively!

In using rice, the way it is cooked is of great importance. The shape of each grain should be preserved; it should swell while cooking and absorb water, but should not break down and form a paste with the other grains. Rice oversoaked with water is not so readily digested, as it cannot be so quickly attacked by the digestive ferments. I therefore advise the following method of cooking: To a given quantity of rice add a similar quantity of hot water and allow all this water to boil away. The rice, whose grains have swelled but

have remained distinct, should then be taken off the fire and seasoned with a little hot fat and salt. Prepared in this way rice is an exquisite dish, of which a lavish use can be made on account of its easy digestion.

Dried vegetable seeds (beans, peas, and lentils), very rich in nitrogen, are also good forms of food when thoroughly mashed and strained,—that is to say, when their cellulose is removed; this is likewise the case with potatoes, Italian alimentary pastes, etc.

Green vegetables are unquestionably of service in insuring the regular working of the large intestine, but as articles of food they are of very doubtful value, being composed mainly of water, cellulose, and chlorophyl. Tubercular patients, who need their entire digestive power for recovery, will do well to use them only in moderation. Even when mashed and thoroughly cooked, mixed with cream, eggs, and salt, they are not nourishing, and they (as do also dried vegetable seeds) have a drawback in that they give rise to great intestinal fermentation and to flatulence that is disagreeable and sometimes painful. It is preferable to seek in the use of bread, potatoes, or possibly carrots, and particularly cereals, the residuum necessary for the regular exoneration of the bowels.

The usefulness of cereals, vegetables, and alimentary pastes depends not only on their nutritive value, ready digestion, and regulating action on the bowels, but on the condition of relative rest in which their free use places the organs appended to the digestive tract. The liver, which acts as a filter on the substances absorbed in the intestine, is supposed to protect the blood from the toxines that may accompany them. Now, these toxines are often produced by fermenting meat with its ptomaines, and nothing is commoner with consumptive patients than to see the liver become enlarged and sensitive, the tongue furred, and the intestines loosened.

No doubt all the abnormal fermentations of the digestive tract, and especially the acid fermentations (among which the acetic fermentation is the chief one), attack the tissue of the liver and lead to sclerosis, steatosis, or the different forms of diffuse hepatitis; but I wish particularly to point out to you the preponderating action in this form of liver congestion of over-indulgence in meat. The danger of an immoderate consumption of meat is a very real one, particularly when the digestive power is below the normal. On the other hand, a vegetable diet has always been considered, and justly so, as

a diet of rest for the stomach, and of all alimentary substances milk is the one that is most efficient as an intestinal disinfectant.

A physician must, therefore, guide his patient through all these different risks in diet, and must always bear in mind that an exclusive regimen is always unadvisable. A mixed dietary with a predominance of nitrogenous food is best suited for consumptives, and the medical adviser's skill will appear in adapting to this ideal regimen a digestive apparatus that is often defective. This duty is frequently rendered still more complicated by a constitutional condition which would seem to forbid any high living,—gout or rheumatism, for instance, which are quite common in tuberculosis. In such cases cereals and vegetables will be of great use, though the fundamental idea of the regimen should not be changed; it must still be mainly nitrogenous, though red meat may be replaced by white, and the raw beef or mutton pulp by cooked chicken pulp.

Desserts, except cheese and fruit baked without butter or sugar, are not advisable for these patients; it will be best to give them up, and replace them by lightly sweetened milk and egg dishes.

As regards the best form of drink, this will depend on the patient's digestion. Red wine will rarely succeed, and in many cases its simple omission from the patient's diet will relieve his dyspepsia. Good spring water, with perhaps a little white wine or beer, is the best drink. When there is some intestinal irritation milk is better still at meals, but in small quantities,—not more than a glass. Malted beer is sometimes of use, and also weak tea or coffee; but this depends on the individualities of each case.

# TREATMENT OF CARBUNCLES AND FURUNCULOSIS BY BEER YEAST.

CLINICAL LECTURE DELIVERED AT BROCA HOSPITAL.

BY L. BROCQ, M.D.,

Physician to the Paris Hospitals.

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Gentlemen,—Towards the end of 1894 one of my patients, who was suffering from a tenacious attack of furunculosis that I had not succeeded in checking, told me one day that for the past two weeks he had been treating himself with beer yeast; that since he had done so no new boils had appeared, while those that were in activity when he began the treatment had healed up with extraordinary rapidity. I must confess that I received this statement with incredulity, as I had never heard of beer yeast as a therapeutic agent; and yet I *ought* to have known of it, as it had at that time already been mentioned in several publications. The patient I just referred to had heard of it while travelling in the north of France, where, having spoken with various persons who had been cured of boils by beer yeast, he had immediately used the treatment for himself with success.

Now let me give you my personal experience with this remedy, both in my own case and with some of my patients.

Since 1892 I have been afflicted every four or five months with a couple of medium-sized carbuncles. These manifestations took the place of frequent attacks of asthma, from which I had suffered since the age of eighteen. From 1891 to 1894 these carbuncles varied in size between the volume of a pigeon's egg and that of a turkey's, and were accompanied by inflammation, lymphangitis, adenitis, œdema, fever, and general disturbance, sometimes even with small abscesses.

In 1894–95, exasperated and worn out by the steady recurrence of these carbuncles, I tried treating myself during the attacks by the extract of colchicum seed, and even published a short note on

this method in February, 1895. But the colchicum gave rise to severe pain in my stomach, and did not seem to me to act as rapidly as the beer yeast did, which I was trying with some of my patients, so that, towards the end of 1895, I determined to try the beer yeast on myself, since which time I have not had a single carbuncle that went through its complete evolution. The following is an instance of what now happens in one of my crises.

On Thursday, January 5, I felt unwell and feverish, while in the region below the chin I noticed the deep and peculiar sensation which every one in the habit of having carbuncles recognizes as the pathognomonic sign of the development of such an inflammation. The following morning I could already feel an intradermic lump the size of a pea, with radiating pains in the neck and general uneasiness, so that I was certain I was threatened with a carbuncle.

On Saturday, the 7th, I began the beer yeast treatment; the painful induration had increased to the size of a small hazel-nut and troubled me a great deal. On Sunday it was as large as a very big hazel-nut, with well-defined limits but without any central head, which for a person with *my* history meant that it was a carbuncle and not a boil. On Monday the infiltration was still larger, forming a clearly visible tumor, with redness of the skin; but a curious change had occurred, as it had become almost entirely painless both spontaneously and to light pressure. Tuesday the condition seemed to be about stationary; still, in the evening the infiltration of the skin appeared to have decreased a little. Wednesday the surrounding infiltration had almost entirely disappeared, and the central lump had also decreased in size. On Thursday the nucleus was no larger than a very small cherry, and I was able to raise my head and even throw it backward without feeling anything more than a slight tension at the diseased spot.

On the following days the improvement continued, so that by Sunday the lump was no larger than a big pea; it was hard, painless, and caused no trouble whatever. The upright collars I wear, and which I had been obliged to keep open during the first days, now rubbed on this lump without making me feel it. This lump remained for three or four weeks, during which time I continued the yeast treatment regularly, as if I stopped the yeast a few days the lump became painful and began to swell again.

Such is the evolution, in my case, of a carbuncle treated from

the outset with beer yeast internally, without the slightest local application, either spray, antiseptic lotion, salve, or plaster. The small lump remains with me for a long time,—in some cases, four, five, or six weeks; perhaps as long as the anthrax would have lasted if it had gone through its entire evolution. I may add that sometimes while I am on the yeast treatment I notice small, pointed lumps appear on my neck, the beginning of boils; but they never develop or grow hard, and are never painful.

When I first began to try the yeast treatment on myself, I commenced its administration only when the carbuncle was in full evolution. I then noticed the following facts very distinctly: (1) On the third day after I began taking the yeast the pain decreased rapidly and disappeared entirely by the fourth day. (2) On the fourth or fifth day the surrounding inflammation, lymphangitis, and œdema disappeared with a most astonishing rapidity. (3) The suppuration decreased noticeably about the fifth day, and the carbuncle, although in full swing when the yeast was begun, seemed to stop short in its evolution.

It is scarcely necessary to say that during the four years that I have known this remedy I have prescribed it to a number of patients. In certainly nine cases out of ten its effect has been good. The number of cases I now have is over fifty, but I will only give you a few details about four others besides my own, which will be quite sufficient to convince you.

The first patient, a man of fifty-eight, from the provinces, had suffered for thirty-five years from constant furunculosis, which had riddled with boils and carbuncles his back, sides, legs, neck, and the upper part of his arms. Nothing appeared to do him much good, and he had resigned himself to antiseptic lotions and Vidal's red plaster, when I put him on beer yeast. Thereupon the scene changed completely. At the end of a week he came to see me entirely transformed, and his skin was clearing up before our very eyes. After two weeks, for the first time in thirty-five years, there was not a boil left. He took the yeast for a month, and then stopped, but three weeks later two boils appeared; he began the yeast again at once, and in three days the boils ceased to increase and then gradually disappeared. Shortly afterwards a few other boils appeared while he was taking the yeast, but they all aborted.

Since then he has been entirely free from boils, carbuncles, and,

what is stranger still, acne, which he had constantly had on his shoulders from the time he was a boy. It is true that in his dread of a recurrence of his boils he has taken the yeast every other month, but he tolerates it admirably and claims that his health has never been better than since he began to take it.

My second patient, a very stout woman of forty-five, had suffered for six months from intense furunculosis, consisting in successive and incessant eruptions of boils of all sizes, some of them almost carbuncles, on all the surface of the body and limbs. Carbolic spraying and antiseptic lotions had produced a painful eczematous eruption, and the patient had tried all the usual methods of treatment. I prescribed the yeast treatment, boric lotions, zinc salve on the lymphangitis and eczema, and zinc plaster on the boils, of which there were about a dozen when I saw the patient for the first time.

When, two weeks later, this lady returned to see me, all the boils had entirely disappeared, and no new ones had shown themselves. Improvement had begun on the fourth day of the yeast treatment, and by the eighth all the boils had aborted. I advised her to keep on with the yeast for another week, and to take to it again at the slightest sign of a boil, in which case she was to report to me again. This was a year ago, and I have not seen her since.

My third patient, a man of fifty-five, had been in the habit, like myself, of having every four or five months a carbuncle of small or medium size on the face or neck. His urine contained neither sugar nor albumen (as was also the case with my other patients). Before he began the yeast treatment the carbuncles were very painful and were as large as a pigeon's egg or a small hen's egg; they suppurated abundantly, were accompanied by considerable œdema, and lasted from four to six weeks.

This man, who also has gravel and is in constant fear of renal complications, only takes the beer yeast under protest; he always waits my positive orders to take it, and consequently only begins his treatment on the fourth, fifth, or sixth day of the carbuncle's evolution, when it has already entirely formed and sometimes even suppurated. But in forty-eight hours, or in three days at the most, the œdema lessens; on the fourth day the carbuncle begins to collapse and the suppuration to become less abundant; while from the sixth to the eighth day the formation of pus ceases altogether and cicatri-

zation takes place. There always remains, however, a small, hard nucleus, which takes weeks to disappear.

The fourth case is a girl of eighteen whom I have taken care of for years for psoriasiform seborrhœa of the axillæ. For several months she had been troubled with small abscesses of the same regions, when finally I prescribed the beer yeast. In ten days the two abscesses that had opened had closed up entirely, three other inflamed points had aborted and disappeared, and there were no new foci. I advised her to continue the yeast for another two weeks, and have since heard through her mother that she has had no further trouble.

These are the most illustrative cases I have to cite to you. I have used the treatment often, both in hospital work and in private practice, but it is not always possible to follow the cases closely enough in order to publish them. Now let me tell you how the beer yeast treatment is administered.

I am in the habit of prescribing fresh beer yeast. The Paris brewers deliver it to me in the form of a light chestnut-colored cream, not unlike a pale chocolate cream. When it is allowed to settle in a glass it separates into three layers: at the bottom is a soft, thickish portion having the color of dark coffee and milk; above that is a layer of a dark chestnut-colored liquid; while on top, by far the thickest layer, is a sort of dense, dark coffee-and-milk colored cream, that ferments with great activity. When I wish to take some I begin by mixing the three layers thoroughly; I then take a full teaspoonful of the liquid and stir it in a wineglassful of ordinary or of an alkaline water. This dose I take three times a day at the beginning of each meal. The taste is not very bad, but the yeast must be quite fresh; as soon as its taste changes and becomes a little sour, I think it is advisable to get a fresh lot. Whenever possible, it is well to get it fresh every day in summer, every other day in winter when the weather is not cold, every three days in really cold weather.

When beer yeast cannot be obtained, it may be replaced by baker's yeast, which is a solid substance, of the consistency of Roquefort cheese, whitish yellow, of which the dose is a lump the size of a hazel-nut mixed in a little water at each meal, or else mixed with equal parts of honey. I am inclined to believe that the stomach stands the beer yeast better, and that this substance is also more active from a therapeutic point of view.

In the north of France the physicians give much larger doses of beer yeast,—as much as two or three tablespoonfuls a day, mixed in beer.

It is probable that with this remedy, as with so many others, there must be great differences between different persons as regards its action, efficacy, and the way it is tolerated. One person will get satisfactory results from a small therapeutic dose, while another will require a much larger amount, so that in some cases we may have to double or even triple the dose that we usually prescribe.

Furthermore, and this is a peculiar point with this treatment, it would seem that different beer yeasts cannot be compared with each other as regards efficacy. This important matter will have to be cleared up by further research, and it would be highly advisable if our chemists could succeed in supplying us with an article whose effect would be constant and unvarying.

This medication has some drawbacks, but, as a rule, if the patient is docile and persevering and the beer yeast fresh, the treatment is easily borne. When intolerance manifests itself it takes the form of heaviness in the gastric region, acid regurgitations that follow each other with most disagreeable frequency and abundance, and diarrhœa, though the latter is quite unusual.

The effect of the treatment on carbuncles I have given above. In the case of boils it is naturally much more rapid. Up to the present time I have not had occasion to try it on a very large carbuncle with really serious complications. In view of the fact that when the treatment is discontinued too soon the carbuncle shows signs of breaking out again, it is advisable to keep the treatment going for quite a while in all cases where the digestive tract does not become intolerant.

I have tried this treatment also in cases of hidrosadenitis of the axilla and in other suppurative dermatoses, such as large, inflammatory acne, chronic acne, staphylococcic folliculitis, and in some kinds of sycosis; but it has seemed to me that its action in these different complaints, although manifest, is less rapid, less evident, and much more uncertain than in furunculosis.

Its effect in furunculosis is so satisfactory that, if I can rely on my own experience in the matter, I should say that fresh beer yeast in furunculosis is as much of a specific as mercury is for syphilis or quinine for malaria. It is only fair to add, however, that two of my

patients claimed they had not been influenced by it in any way, that others said they simply could not take it at all, while still others said it only acted after some time. In such cases the unwillingness and lack of perseverance on the part of the patient, no doubt, plays a certain *rôle;* but still, in the presence of such failures, which have also occurred in the practice of other physicians, it is well to study the difference between yeasts, the effective dose for different patients, etc. Certainly in my hands this treatment has proved preferable to all its predecessors: benzonaphtol, boric acid, camphorated tar, sulphur, hyposulphite of soda, colchicum, etc., and it acts by itself, without any complicated and annoying bandages or dressings.

# TREATMENT OF SOME OF THE MORE COMMON FRACTURES.

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In the treatment of a fracture the first thing to be taken into consideration is reposition of the fractured parts, and this should be done as early as possible. If the patient is in favorable surroundings, it is advisable that the fracture be reduced just as if it was the intention of the surgeon to leave it permanently. If, however, it is necessary to transport the patient to some other locality, a temporary dressing, such as will prevent motion of the fractured parts, is the proper step; then, after arriving at the patient's final destination, complete reduction should be accomplished without delay, and a proper dressing applied.

As a first step in the treatment it is important that the diagnosis should be completely settled, under anæsthesia if necessary, and while the patient is under the influence of the anæsthetic replacement of the fragments may be accomplished, and a permanent fixation dressing by means of splints or otherwise should be effected. Plaster dressings, as a rule, are not advisable in the earlier stages of fracture, as if they are then put on they may become too tight after swelling takes place, and not only cause the patient a great deal of pain and some risk, but usually require removal. It is true that a plaster-of-Paris dressing put on in the early stages over a liberal amount of absorbent cotton may be split open after it has set, and used as a case to fix permanently the fractured ends of the bone with considerable satisfaction. It can be well understood, for instance, if there is a fracture of the humerus, and a plaster dressing is applied over an abundant supply of absorbent cotton and allowed to set, then cut so as to make a case fastened in place with bandages over the outside, that it produces a very satisfactory fixation of the frac-

tured ends of the bone. This form of case may be more or less conveniently removed and the fracture inspected.

As a rule, however, I think it is more satisfactory to fix these fractures with ordinary splints for the first seven or eight days, when the splints may be removed and a plaster dressing applied if it is thought advisable. It must be remembered that only a limited amount of repair takes place in the first six or eight days, except in a few cases, and that if the fracture is properly set at once the fragments will not be displaced except by considerable violence after three or four days, because of lymph which is poured out around it fixing it in place, and consequently a limited amount of motion will not disarrange it. For this reason fractures may be safely re-dressed in four or five days without risk of displacement of the fragments if they are carefully handled. But it is also to be borne in mind that fractures which are improperly set, or which have become displaced by any kind of violence, can very readily be re-set at any time within the first five or six days after the fracture has occurred, without exercising a great deal of force and without more than bearable pain to the patient; that is, the union which has formed is not firm enough to necessitate much force in replacing the fragments. The occasion of most of the displacement which occurs after fracture is muscular violence due to irritation communicated to the muscles because of the traumatism. This muscular irritability usually ceases in the course of from thirty-six to forty-eight hours, and in re-dressing, unless there is much disturbance, muscular contraction does not occur, and consequently displacement from this source is not to be feared.

As soon as a bone is broken, contraction of the muscles displaces the fragments. The contraction continues even after the fracture is reduced, and is only overcome by splints; but after thirty-six or forty-eight hours this muscular contraction does not occur, and re-dressing may be safely done. For this reason fractures may be inspected much more satisfactorily where simply a movable dressing has been applied primarily. This is one objection to the primary fixed dressing,—it renders inspection of the fractured parts impossible. In addition to this, there is the further objection already mentioned,—the danger of swelling and the necessity of removing the plaster dressing. Again, the plaster dressing if applied while the limb is swollen will become loose at the end of ten or twelve

days by subsidence of the swelling. These objections make it seem to me wise to apply a simple dressing to most fractures for the first six or eight days, after which those that seem to require it may be fixed in plaster of Paris.

Plaster dressings are allowed to remain in place for a variable length of time, depending upon the character of the fracture.

Fractures of the inferior maxillary bone require to be carefully protected for a period of about three weeks, after which the bone may be allowed more or less freedom; up to this time it must be immovably fixed.

Fractures of the radius and ulna, especially in children, require splints only for about three weeks, and even in the adult it is advisable to remove the splints between the fourteenth and twenty-first day, so as to allow the patient to acquire the use of the muscles and prevent atrophy, on the one hand, and injury to the superficial nerves that occupy this region of the body from plastic deposits, on the other. In fractures of the humerus splints are required to be left on for the period of a month, and in fractures of the femur for a period of eight to ten weeks. Rarely earlier than at the end of eight weeks is it safe to remove the dressings in fractures of the femur in any situation except in intracapsular fractures. Fractures of the lower part of the tibia require dressing for five or six weeks. If plaster dressing is applied, it should be left for a period varying from four to six weeks, depending upon the age and vitality of the patient. The same rule applies to splints. Where there is a fracture of the fibula alone, the dressing may oftentimes be removed much earlier, because the fibula really acts as a support to the leg, a point of attachment for the muscles, the tibia practically receiving all the weight of the body, and patients are able to go about on a crutch in fracture of the fibula soon after the dressing has been applied.

What is known as the ambulant treatment of fractures, as applied by a number of surgeons, enables the patient to get about with a fracture of the lower extremity at a much earlier period than under the ordinary plans of treatment. Various methods have been devised for supporting the leg in such a way as to enable the patient to bear a certain amount of weight on it and get about on crutches. This apparatus, usually a splint or some other device, is used in fractures of the lower leg. Fractures of the femur usually require a little different treatment from fractures of the lower leg. I am inclined to think

that a certain amount of irritability which is in this manner communicated to the point of fracture aids in repair and prevents, perhaps, failure in union in a certain proportion of the cases under ambulant treatment; but only in people with strong nerves, with much power of resistance and courage, is it possible to do much with this method of treatment. It is astonishing how many people, who are apparently doing well for the first three weeks after fracture of the lower leg, will be frightened at the idea of being obliged to get up on crutches. It does not occur to them that they can bear any weight on the leg, because of the pain they feel, which is due to interference with the surrounding structures by the plastic exudate which is thrown out at the point of fracture. Because of the pain which they experience, they are afraid at first to bear their weight on the fractured leg. We know, however, that in the vast majority of cases of fracture of the lower extremities there is complete union at the end of three weeks, but there remains a certain amount of tenderness and pain on motion because of the plastic exudate. For this reason the plan of urging the patient to take exercise by the ambulant treatment in the first ten days after fracture must be applicable only to a few individuals.

We now come to the consideration of special fractures. Fractures of the cranium are of more importance with respect to injury and dangers to the soft parts than to any injury of the bone; consequently I have always described fracture of the cranium especially with the view of calling attention to the regional surgery of the brain itself.

Fracture of the malar bone is a rare accident, and is liable to occur only in direct violence of such a character as to be of more importance from the injury it communicates to the soft parts and adjacent structures, perhaps, than to the fracture of the bone itself. The zygomatic process is occasionally broken by violence of this kind, and the bones may be markedly displaced. They can usually be reduced by inserting underneath the fractured portion a strongly curved needle which carries a heavy silver wire or a piece of silk with which the wire is drawn through. This is a suggestion originally made by Matas, of New Orleans, and is a very satisfactory means of raising up the zygomatic process when depressed. Fractures of the malar bone proper are usually treated by local applications of a sedative character to allay the inflammation of the soft parts, and if there

is any special displacement it is replaced as the judgment of the surgeon indicates.

Fracture of the superior maxillary bone is also a rare accident, and is usually due to sudden violence. It is not infrequently attended by such severe injuries to the brain as to result in early death. There are no special indications for its treatment, except perhaps where the alveolar process has been broken off, and such fractures should be treated according to the judgment of the surgeon in charge. No special rules can be laid down in the treatment of injuries of this kind.

Fracture of the nasal bone is quite a common accident. It is usually due to direct violence; it may be the result of a blow, or the passage of some heavy weight over the nasal bones. It may be caused by something falling from a considerable height and striking upon the face, but in such cases other severe injuries also usually occur. The injury is generally attended with profuse hemorrhage from the nose, which in a majority of cases can be readily controlled by ordinary measures. Fracture of the nasal bones is one of the injuries in which replacement of the fragments must be accomplished very early; if it is postponed for thirty-six hours, there is great difficulty in replacing the parts; not only this, but, because of more or less contusion of the soft parts, manipulation is usually accompanied by considerable hemorrhage. Generally speaking, if the patient is seen early, the fractured portions of the nasal bone can readily be replaced. The difficulty of holding them in place, however, is sometimes considerable. This is perhaps best done by using the ordinary harelip pins. Occasionally it may be necessary to use a little stronger or firmer material. My experience has been, however, that by the use of the transfixing pins at the base of the fracture, the fragments can usually be held in place. This pin is pushed completely through the nose, so as to transfix the nasal bones at their base and hold them well up into place. It is advisable sometimes to pass the pin directly through, sometimes obliquely in proportion to the situation of the fracture. If the vomer has been fractured and the displacement is to one side, it can usually be held firmly in place by passing the pin in a horizontal direction through the cartilage and up through the body of the vomer. The pin is introduced at the margin of the cartilage and passed directly upward through the vomer in such a way as to bring it into its natural position. The pin can be removed in from

forty-eight to sixty hours. It is of the greatest importance to overcome displacements occasioned by injuries in this region, on account of the deformity that a crook in the nose entails. The least deflection of the nose to one side externally produces a very marked deformity, which it is always desirable to avoid.

Fracture of the inferior maxillary is a common accident. It may occur upon one side or both, and it may be either simple or compound. In the great majority of cases of fracture of the lower jaw that have come under my observation, there has been a tearing of the mucous membrane connected with it, constituting a compound lesion. This is a complication sometimes giving trouble; still it is a fact that fractures of the inferior maxillary bone usually do well. Fractures which are upon one side only, and those in which there is no injury communicating with the mouth internally, do exceptionally well under a simple dressing. This dressing consists in the application of a piece of ordinary pasteboard, or of the tin splint suggested by Levis, which is passed directly over the maxillary bone and made to fit perfectly, and is held in place by a bandage or an apparatus suggested by Hamilton. Hamilton's bandage is made of leather cut so as to fit over the chin, starting it underneath and allowing it to extend well to the outer side and in front; the leather is wet and moulded to the chin closely enough to hold the fractured bone in apposition. This apparatus is mentioned in most text-books, and it is a very satisfactory method of treatment of fractures of the inferior maxillary bone. Double fractures in this situation necessarily require wiring by some method until union takes place. The fragments may be simply fastened together by the teeth upon each side of the fracture with ordinary silkworm gut, silver wire, or even silk. In fractures of the inferior maxillary bone which are already compound, which are double, or which consist of three or more fragments, the ones upon each side of the central fragment usually require some other means than the simple application of a supporting bandage or splint. However, if the fragments are so situated as to be held by the application of a silver wire or silkworm gut between the teeth, very satisfactory results may be obtained by this method and the use of the Hamilton apparatus or the ordinary tin splint of Levis. These fractures require to be carefully watched, and the mouth should be kept clean by the use of some antiseptic. Any one of the various forms of mouth-washes or antiseptics will answer the purpose, and probably listerine diluted

with water is the one most commonly used. The mouth should be thoroughly cleansed three or four times a day, or oftener if necessary. It is a fact that compound fractures in this region give less trouble than compound fractures elsewhere. They usually heal in the course of three or four weeks, without much suppuration, if the mouth is kept well cleansed and infection remains under control.

Fracture of the clavicle is probably the most common of all fractures except that of the radius. Fracture may occur in any portion of the clavicle, but is most common in its middle or outer third. It is very frequent in children. It is not uncommonly overlooked, because usually the pain and discomfort apparent in the child, especially if too young to give much description of the symptoms, are usually referred to the arm and shoulder, and the attending surgeon, having examined the shoulder, humerus, etc., and finding no evidence of injury there, in not a few instances has been known to pronounce the child entirely without injury, when fracture of the clavicle existed and escaped his observation because especial attention was not drawn to it. The diagnosis can usually be made if sought for. Even if there is no displacement, pressure along the line of the clavicle will elicit pain and tenderness. In all injuries of the child or even the adult coming under my observation, if pain is referred to the shoulder and down the arm, I make it a rule to pass my fingers along the region of the clavicle in order to determine if it be the seat of injury, even though other injuries are found. It is easy enough to examine the clavicle by simply passing your fingers along its course to determine whether there is any pain or displacement. The displacement is almost always in the line of the shaft. The fragments lie along by the side of one another, either antero-posteriorly or laterally. Most commonly the displacement is lateral, one fragment lying above or behind the other. Reduction of these fragments is usually easy enough, but to maintain them in proper apposition is more or less difficult; in fact, complete prevention of future displacement is almost impossible in most cases. Generally speaking, the prognosis of fracture of the clavicle is favorable, with complete restoration of the function of the arm. There may be a little shortening, but not sufficient to cause any considerable deformity, and the function of the arm is completely restored. In girls and young ladies it is oftentimes a point especially desired to overcome or prevent any deformity, from an æsthetic stand-point; so especial care should be exercised in

the treatment of fracture of the clavicle in such cases. Rest in bed, absolute recumbency, and careful application of compresses and splints will oftentimes prevent deformity; but patients are seldom willing to undergo complete confinement to bed for two or three weeks because of a simple fracture of the clavicle.

The usual treatment of fracture of the clavicle consists in one of three dressings. Perhaps the simplest of all is the method suggested by Sayre, which consists in fixing the arm at the elbow, and then passing a strip of adhesive plaster two and a half inches in width around the waist, fastening it just above the elbow (passing it around the arm once), so as to hold the arm backward, and by dressing the arm back in this way, by putting the clavicle on the stretch, pressing the deformity out. In order to overcome this displacement backward, another strip of adhesive plaster is split to allow the olecranon process to slip through it, and then, the arm held upon the side, the strip of adhesive plaster is carried directly over it in front and behind around the opposite shoulder, when the strips meet. This presses the arm forward, the other strip holds it firmly in this position, and the result is that the deformity occasioned by the displaced ends of bone is almost completely overcome. Perhaps as satisfactory results are obtained in this way as in any other.

The second method is one suggested by Moore, of Rochester, New York, in which a bandage is placed around the elbow in the form of a figure-of-eight, then carried upward over the fractured clavicle and back around underneath the arm upon the opposite side, and then the two ends are fastened together at the back. The arm is then carried in a sling. This is a very simple method of dressing fracture of the clavicle, and in the majority of cases answers as well as any other. In children it is usually advisable to put on some form of dressing which will hold the child's arm steady, and the ordinary Velpeau bandage makes a very satisfactory dressing for this purpose. This, however, does not so easily overcome the deformity as the other methods. In any one of these dressings which holds the bare arm next the skin, it is advisable first to grease the skin with a little vaseline, otherwise the heat occasioned by bringing together the surface of the arm and the body will cause vesication and make inflammatory blisters for the course of five or six days, especially in hot weather. As an additional aid in preventing deformity it is well to put a pad in the axilla, which tends to hold the shoulder and arm out from the

body and in this manner increases the leverage on the ends of the fractured clavicle. This pad should be small in size, and in warm weather it should be dusted with some of the ordinary antiseptic powders, face powder, or borated talcum, or else should be greased with vaseline, to prevent irritation from its presence. The Velpeau bandage is very satisfactory for the treatment of fracture of the clavicle in children. Fracture of the clavicle usually requires the application of a dressing for the period of three weeks, after which the arm may be carried in a sling for a short time, until firm union has taken place.

Fracture of the sternum is usually caused by very severe violence, and most commonly occasions more danger from injury to the soft parts than from displacement of the fragments. If the fragments can be seen positively displaced, they should be reduced if possible, or may be dressed in that position if the surgeon can do so by simple manipulation. If this cannot be done, it would appear that the indications are such as to require some operative treatment,—that is, if the patient is not injured beyond the possibility of recovery by reason of violence to the soft parts. Simple incision down upon the sternum and the use of either a hook or a tenaculum, or a small gimlet bored into the sternum, may aid in replacement. This is not a common accident, and in the majority of cases requires little or no attention. I have seen but one case of fracture of the sternum, and that occurred in connection with fracture of the spine. There was marked dislocation of the fractured portions of the spine, so as to draw the fractured portion of the sternum inward, the spine being dislocated outward. The condition was such that it was utterly useless to attempt anything. The patient continued in a condition of complete paralysis for five weeks and then died.

Fracture of the ensiform cartilage occasionally occurs, the cartilage being turned downward so as to press upon the soft parts and interfere with the stomach in such a way as to require replacement. This may usually be accomplished by pressure with the fingers, and when once replaced the fragments ordinarily remain in position.

Fracture of the ribs is a very common accident, and is generally the result either of the passage of some heavy weight over the body or of falling from a considerable height. It is always due to direct violence. Indirect violence can scarcely occasion fracture of the ribs, or, if it did, it would be attended with so much injury to other

parts of the body as to make this accident comparatively unimportant. It is not uncommon that a number of ribs are fractured by the same act of violence, two, three, or even more. If there is much displacement there is liable to be injury to the pleura, and occasionally there is also injury to the lung. Bloody expectoration indicates injury to the lung, perhaps by puncture by the fractured end of a rib into the lung structure itself. Of course bloody expectoration may result from violence and contusion without puncture. The production of emphysema, or the escape of air externally underneath the cellular tissues, indicates puncture of the pleura, with injury to the lung itself. Occasionally when the fractured ends of a rib penetrate the pleura or the lung structure, a great deal of pain is felt by the patient upon motion, and the accident is also complicated by considerable coughing. Impeded respiration, cough, violent pain, etc., indicate that the fractured end of the rib has penetrated the pleura and perhaps the lung tissue. Under these circumstances removal of the foreign body is imperative. If it cannot be done by manipulation, it is necessary to cut down and make a compound fracture, opening well into the tissue and removing the fractured end of the rib, if necessary, by resection. This is not a very common accident, but has happened in my experience twice.

Ordinarily fracture of a rib produces merely the symptoms of contusion. The patient complains of pain upon respiration and usually upon pressure or movement. With the patient lying on his back, by pressure on the sternum, which depresses the ribs, the point of fracture, if one exists, can generally be determined. Pain accompanying this manipulation, if constantly referred to a point on a rib, is almost a pathognomonic sign of fracture of a rib. Displacement is not nearly so common in fracture of the ribs as in fractures of other bones of the body, because the attachments of the rib act as a support to hold it in place. In a certain proportion of cases, however, the finger passed over the seat of fracture will detect more or less displacement of the broken ends. Nearly always pressure over the rib at the site of the fracture will indicate its presence by the pain thus caused.

Unless there is marked displacement, or some injury of the pleura or lung, fracture of the ribs usually requires but little treatment. Fixation of the affected side with broad strips of adhesive plaster is the favorite treatment of a number of surgeons. Adhesive plaster

remaining for a considerable time in contact with the skin, especially in warm weather and in warm climates, is liable to occasion more or less vesication and is, I think, a less satisfactory method of treatment than the application of a broad bandage, which more or less limits thoracic respiration and obliges the patient to breathe chiefly with the diaphragm and muscles of the abdomen. It is my custom to take a bandage eight or ten inches wide and sufficiently long to go twice around the body so as to get firm compression of the thorax, then pass this around the patient and draw it tightly enough to restrict the movements of the thorax, making it as tight as is comfortable to the patient. A compress may be applied over the seat of the fracture if it be deemed necessary. This bandage should be fixed in place by safety-pins, and the amount of pressure it exerts can be graduated at any time by the attendants. It may be readily understood that the compression made by the bandage passing twice around the body can be easily regulated without any considerable tension upon the pins, and the bandage, moreover, cannot slip. All of these features cannot be obtained with a bandage which passes only once around the body.

Usually fractures of the rib heal in from fifteen to twenty days, when either the bandage can be removed or the amount of pressure that it exerts can be gradually lessened.

The contusions which usually accompany fracture of the ribs may frequently make the duration of recovery considerably longer. The effusion of blood underneath the skin sometimes makes it look as if some serious injury had been communicated to the soft parts, and oftentimes greatly alarms the family and the patient. This discoloration ordinarily does not appear before the third or fourth day after the injury. It appears, however, much earlier in contusions in this situation than it does in the more fleshy parts of the body, because of the superficial location of the blood-vessels. If there be any evidence of serious hemorrhage occurring after fracture of this kind, rupture of the intercostal artery must be borne in mind, with possibly the escape of blood into the pleural cavity. Under these circumstances an exploration should be made at once and a ligature applied to the wounded vessel.

# Medicine.

## PROGRESSIVE PERNICIOUS ANÆMIA.[1]

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF BERLIN.[2]

BY PROFESSOR ERNST GRAWITZ,

Docent of Special Pathology and Therapy at the University, and Director of the Hospital Charlottenburg, Berlin, Germany.

Gentlemen,—Our patient has, as you see, most of the classical symptoms of that disease which has attracted so much attention of late years,—progressive pernicious anæmia. It is the second form of essential anæmia, chlorosis, the other form, having been the subject of our last lecture. The disease received its name from Biermer, the late director of the clinic at Breslau, who described a series of cases of it in 1868. He had been at Zurich, and it was in Switzerland, where the disease is more common than elsewhere, that most of his cases had been observed.

As is the case in chlorosis, most of those attacked are women, though there are not many other similarities between the two diseases. The external appearances of sufferers from the two diseases are very different. The extreme, almost waxen pallor of those suffering from progressive pernicious anæmia has none of the yellowish-green tint that characterizes chlorosis, and from which the disease is so well named. Chlorosis attacks, by preference, during the developmental period, from fourteen to twenty, and pernicious anæmia between the ages of twenty to forty, though many of the cases occur in the third decade of life. In neither of the diseases does the subcutaneous fat disappear, which is surprising enough, con-

[1] Professor Grawitz asks us to state that the subject of this clinic is "Pernicious Anæmia in Adults." "Pernicious Anæmia in Children" is a special chapter by itself

[2] Reported by James J. Walsh, Ph.D., M.D.

sidering the weakness and want of appetite so marked in both the disorders.

Usually the first symptom of pernicious anæmia that is noticed by the patient is an intense feeling of physical and mental weakness. There is a constant tired feeling, associated with profound psychic apathy. So little does their expression change in severe cases that a suspicion of melancholia may be aroused, the patients being so absolutely impassable. The speech also may become drawling. It is slowness of mental and physical processes, however, not hallucination, that is present. Defective metabolism has rendered cellular activity tardy. The patients' weakness is sometimes so great that they may fall back exhausted during the physician's examination, and the taking of the history becomes a difficult task because of the slowness and incompleteness of mental processes.

Sometimes the first symptom the patient notices is a tendency to dropsy, which occurs especially in the legs, though, of course, it usually indicates an advanced hydræmic condition of the blood. There will often be in such cases a suspicion of kidney disease, and, as albumen may be found in the urine and changes found in the eye-ground on ophthalmoscopic examination, the examination of the blood will have to be the deciding factor. I cannot insist too much on the importance of ophthalmoscopic investigation in all cases of anæmia, as it often gives the clearest evidence of the pathological process that is at work.

Besides the hemorrhages in the retina, there may be a series of subcutaneous or submucous effusions of blood, but these occur late in the disease, usually *sub finem vitæ.*

Loss of appetite is often a most striking feature. It is complete, and rapidly leads to exhaustion from inanition, though emaciation may not be marked. Where some appetite remains symptoms of indigestion often develop, and there is usually chronic constipation, which may, however, be replaced by troublesome diarrhœa. In all cases where the temperature is carefully taken febrile movements are noted. The whole set of clinical symptoms is especially characterized by being almost inevitably progressive. Long remissions may occur, but the symptoms recur. Biermer lost all of the cases that he originally described, and, while the prognosis of the disease has in recent times become less unfavorable, the term progressive is still very properly applicable.

Despite the immense amount that has been written about the disease of late, its pathogenesis is still a riddle. The pathologists have spared no efforts, no organ, however unimportant it might seem, has been left unexamined, to find a pathological basis for the disease, but in vain. In certain cases, with a similar blood picture and the highly anæmic condition found in pernicious anæmia, certain organic changes have been found. Notably has this been the case for the severe anæmia that accompanies the cachexia of carcinomatosis, generalized carcinoma metastasis, such as is sometimes known to occur after cancer of the stomach. These cases have, however, been carefully separated, and the criterion given originally by Biermer and adopted by Eichhorst still holds good. There are fatal anæmias in which no organic change is found to account for the condition, and these are the only ones to which the name progressive pernicious anæmia properly applies.

For a time it was thought that certain atrophic changes in the stomach and intestinal mucous membrane might be the pathological basis of the disease, but this atrophy of ventricular and intestinal glands, that Ewald has called anadenia, is found in nearly all severe anæmias and is a secondary change, an effect of the anæmia, not its cause.

In general the organs post-mortem show the effects of the intense anæmia. They are strikingly pale. The heart muscle is of a very pale red, with spots of yellow in it here and there where fatty degeneration has taken place,—the so-called "tiger heart" of anæmia cases. All the muscles have an intense pallor. Only two organs usually stand out, by contrast, a bright red,—the bone marrow, in which the yellow fat marrow of the shafts of the long bones has become of a fresh blood-color and reverted to the lymphoid condition of embryonal life, and the spleen, which in most cases, though not all, is also bright red. In certain cases the spleen is also enlarged, and this condition has been named the splenic form of the disease.

A set of changes that in recent years have attracted a good deal of attention are those occasioned by old syphilis. The smooth, shining base of the tongue, the contracted, scar-like, irregular, sclerotic indentations of kidneys and spleen, and the cicatricial tissue in the testes, that point to an old syphilitic infection that has run its course, are often found. There is also a sclerosis of bone tissue, especially of the spongiosa, that is often very striking, and that I

cannot help but think plays an important etiological *rôle* in many cases of the disease.

Of late years it has been found that various intestinal parasites, especially the ankylostomum duodenale and the bothriocephalus latus, set up by their presence a condition that resembles pernicious anæmia very much. Instead of an essential anæmia in such cases, it is of course evident that we have to do with a secondary anæmia, whose symptoms depend either on the loss of blood occasioned by the parasite in securing its nutrition, or on the absorption of certain toxic albumoses which are the result of metabolic processes in the parasite, or on both these factors.

One thing more pathological investigation has shown. Quincke demonstrated that there was present in the liver, bone marrow, and spleen of fatal cases much more than the usual amounts of iron, showing that more red blood-cells than usual were destroyed in these organs, and the iron of their hæmoglobin had been deposited there.

The tissue that is constantly found in a pathological state is the blood, but it seems clear, even from what I have said, that there is no real unity to the disease, but that the symptom complex may have a number of causes. The essence of the disease is a destruction of red blood-cells, until at last the blood-making organs become exhausted, refuse to respond to the call for new cells, the blood composition suffers in all respects from organic malnutrition, and so the morbid process becomes fatally progressive. At times it would seem as though the blood-making organs themselves were primarily inhibited in their activity, and the red blood-corpuscles, probably of but ephemeral duration, since they have no nuclei, grow ever fewer and fewer. The irritation of the impoverished blood on the blood-making organs serves only to arouse them to the formation of the abortive red cells, the macrocytes and microcytes, the nucleated red cells and the poikilocytes, which form so characteristic a part of the blood picture of pernicious anæmia.

The causes which produce this destruction of red cells and the inhibition of blood-making organs may be summed up as follows. One of the most prominent groups of symptoms is the gastro-intestinal. The loss of appetite and the constipation point to digestive disturbances. There is, consequent upon the long retention and decomposition of imperfectly digested material, a production of toxic substances, which, absorbed by the portal blood, lead to diminished

vitality, even to destruction of red blood-cells. Sir William Hunter showed but a short time ago that auto-intoxication might lead to such effects on the blood. It is in women particularly that this stagnation of intestinal contents is noted; hence their much greater liability to pernicious anæmia.

In the better classes this coprostasis and the decomposition especially of albuminous products which it involves give rise to either blood or nervous disturbances. The albumen molecule is extremely unstable, and this makes it eminently suitable for metabolic purposes and its conversion into toxic substances very easy under unfavorable circumstances. These toxines disturb either one of the two most delicate mechanisms with which they come in contact, the blood-making or the nervous system. Why it should be the one and not the other in any given case it is not easy to say, but either anæmia or hysteria develops.

The presence of indican in the urine shows that there is increased albuminous decomposition in the intestines. While not toxic itself, it is the index of the presence of toxic substances in the circulation. I have found it being excreted in large amounts for months in some of these cases. Sandoz showed that in many cases of severe anæmia the washing out of the stomach and intestines led to prompt improvement, showing that the removal of toxic substances already formed but not yet absorbed put an end to the progressive character of the disease and gave nature a chance to react.

As the result of the presence of these toxines and the gradual impoverishment of the blood, the glandular mechanism of the stomach and intestines atrophies; and this further acts to impoverish the blood by interfering with the secretion of products for digestive purposes, and disturbing the absorptive parts of the mucous membrane. It is a vicious circle in pathogenesis, each defect acting for itself and reacting on the others to make the general condition worse. In women I am sure that these conditions play an important part. When we find that pregnancy is a prominent factor in the disease, we realize that here there is apt to be most obstinate constipation, that the hydræmic condition of the blood during pregnancy still further predisposes to instability of blood composition, and, from lowered nutrition, predisposes the blood-making organs to further disturbances of function, and finally even to organic change.

Next to auto-intoxication defective hygiene is the most impor-

tant etiological factor. By the term defective hygiene I mean any habitual series of unhealthy influences to which the individual may be exposed. In the poorer classes they consist of overwork, with poor, unsuitable, and insufficient food, life in damp, dark cellars or in badly ventilated living-rooms, in the midst of cares and worry, family misfortunes, and sometimes even abuse and injury. In the better classes, especially among the women, it is the desire to imitate the men in everything that of late has invaded so many female breasts. They undertake mental work of the severest kind, take up athletic sports passionately and with a want of moderation characteristically feminine. They write, they ride, they assume wearisome social obligations, interest themselves in charities and charitable organizations unnumbered, and in the midst of it all eat most capriciously and unsuitably. The result is, as I have said, anæmia or hysteria.

That continued malnutrition will produce anæmia I have demonstrated myself experimentally by living for some days on insufficient food, and finding that the blood became disturbed in several ways, but especially in the lowering of the number of red cells. Suppose this continued a considerable time, especially in women, in whom the equilibrium of blood composition is less stable, by a provision of nature to meet the varying calls made upon it by menstrual losses and by the periodical occurrence of pregnancy and its hydræmia, with the often serious loss of blood at the end of it; and it is not difficult to understand that the blood-making organs would finally refuse to react to stimuli, and the blood composition would degenerate.

A third cause which we have learned to think more of in recent years is found in the changes left in the tissues by syphilis. Especially is this true of the bone changes of syphilis, the sclerosis of bone that occurs and seems to interfere with the function of that important blood-making organ, the bone marrow. I was able to demonstrate by means of the Röntgen rays in one fatal case of pernicious anæmia these sclerotic changes, resulting in eburnation, even during life. The changes need not always be as coarse as those observed in that case and yet be sufficient to produce the progressive anæmia. Osteosarcoma may, by a similar pathological process, produce a like set of symptoms. For the investigation of the etiology of certain forms of pernicious anæmia the bone changes constitute, in my

opinion, the most promising field that remains open to us at present.

A fourth source of pernicious anæmia, pointed out by Eichhorst, is a series of persistent, successive, though perhaps slight hemorrhages. The blood-making organs seem to become exhausted and refuse to respond to the stimulus that should lead to the formation of new blood. The simple secondary anæmia that at first results from mere loss of blood becomes a persistent, progressive condition. The blood takes on that essential character of pernicious anæmia, the incapability of proper regeneration. This seems especially to be true in women. They endure single severe hemorrhages better than men, but they do not resist the effect of frequently repeated slight hemorrhages nearly so well.

A fifth cause or series of causes connected with pernicious anæmia is chronic exposure to the influence of poison. One of my own cases, for instance, was an ironer, who breathed and rebreathed during the day the carbonic oxide and other poisonous vapors resulting from the incomplete combustion in the heating apparatus. Others, among them Dr. Hanseman, have found long-standing morphinism as a causal element, and exposure to lead has been thought to have the same effect.

The specific peculiarity of the disease—that which, though it may be produced by any one or a combination of these causes, makes the disease a unity despite multiplicity of causes—is that a pathological condition of the blood develops which no longer permits of a return to the normal. It is, as I have pointed out, a disease of metabolism. Certain functions of the blood, especially that of constant regeneration, are lost. The blood as a tissue ceases to fulfil its physiological purpose and takes on a premature tendency towards dissolution.

Pernicious anæmia looked at as a fault of metabolism, like obesity or diabetes mellitus, makes us realize better its unfavorable prognosis. Diabetes mellitus is not cured, even though by dieting we make the sugar disappear from the urine. By keeping the amount of sugar introduced into the organism below the amount it is able to metabolize, we can absolutely prevent the excretion of the sugar in the urine, but the metabolic disease remains, ready to assert itself as soon as the amount of sugar consumed passes a certain limit, and even in certain cases with a tendency to become spontaneously worse. The obese have acquired the pathological habit of converting even

the albumen of their food into fat, and so, despite all dietary precautions, polysarcia advances. A wrong direction has been given to nature's energies in these conditions, and, even though the causes that originally produced the error of metabolism be removed, the pathological condition itself may persist. So it is with pernicious anæmia: we may stop the insufficient nutrition, the overwork, the continued losses of blood, and the chronic intoxication that occasioned it, but the disease itself continues. It is this property that makes it deserve the name pernicious.

Usually, when pernicious anæmia is spoken of, we think of intense alterations in the size, shape, and number of the red blood-corpuscles, and this is undoubtedly its most prominent feature. At times some of the red cells are so altered in size and shape that it is practically impossible to tell whether they are red cells or not. The blood is, however, altered in all its components. Its hæmoglobin is constantly reduced, not only absolutely but also in proportion to the number of red blood-corpuscles. It is sometimes said that the amount of hæmoglobin in individual red cells is proportionately above the normal, but this is never true and is an impossible condition.

Besides other changes, the protoplasm of the red cells is so altered that it does not take aniline dyes as it does normally. The staining is pale and strikingly uneven. The solid substance of the cells has lost in density.

All the other blood qualities are also altered. Its specific gravity is reduced to 1025 in the severer cases, while the specific gravity of the blood-serum alone is normally 1029. In the same way the quantity of the dry residue of the blood is reduced to from 9 to 10 per cent., while the dried residue of the serum alone is normally $10\frac{1}{2}$ per cent. This hydræmic condition of the blood is very marked, and, when a certain amount of it is diluted with water and compared with a corresponding dilution of normal blood, the reduction in hæmoglobin is very noticeable. Though the ordinary methods of hæmoglobinometry are inexact and therefore of but little scientific value, they also serve to show in a coarse way that the hæmoglobin is reduced.

As the result of the hydræmia patients bleed easily, from even so slight a wound as a pin puncture, and, as in our patient a few minutes ago, we have to take some such means as putting a piece of cotton over it to stop it.

After centrifugation of normal blood with the hæmatocrite, the

proportion of corpuscles to plasma is about even,—*i.e.*, the corpuscular sediment represents one-half the height of the column of material in the tube. In pernicious anæmia, after centrifugation, there may be but a small layer of cells, not more than one-tenth of the height of the plasma,—*i.e.*, from 50 per cent. the cells may be reduced to as low as 10 per cent. of the blood constituents.

The blood-cells themselves are very much altered, but the plasma is also altered, and the use of these various hæmometric methods gives us a good idea of how the blood has degenerated in every way. The red cells have, however, suffered so much more than the serum that the natural conclusion is that there must be a special poison acting for their destruction, while the plasma suffers only from the deterioration of the general circulation.

There is a distinct selective hurtful agent at work, for the other blood constituents do not suffer at all proportionately to the red cells. There are some pathological changes in the leucocytes, but they are unimportant and are evidently due to the general malnutrition. The difference between pernicious anæmia and cancer cachexia, in which the blood suffers as a whole and all its constituents alike are affected, is very striking. In cachectic cases a very striking symptom is always the emaciation, while in pernicious anæmia the subcutaneous fat remains.

A very interesting analogy with the poisonous influence of autointoxication is the fact that, where anæmia is induced by the presence of such parasites as the ankylostomum duodenale or bothriocephalus latus, it is not due alone, nor indeed so much, to the loss of blood as to the absorption of toxic substances produced by the vital metabolism of the parasites. So it is that in these cases it is the red blood-cells especially that suffer much more than the general composition of the blood.

The prognosis of a case presenting the features of pernicious anæmia is not so absolutely unfavorable as used to be thought. Of eleven cases that I have had under my care in recent years where the natural diagnosis would be pernicious anæmia only four have proved fatal. The prognosis depends on the extent of the blood changes. Where the disintegration of red blood-cells is marked, the poikilocytosis extreme, and the reds have lost to a great extent their original disk form, the prognosis is naturally bad.

Ehrlich attributed a specially unfavorable prognostic significance

to megaloblasts, the large nucleated red cells that occur normally in embryonic blood; but I have not found them so significant. I saw a fatal case in which they were not to be found, and in another case, in which they were readily demonstrable, improvement set in which has lasted up to the present time and the patient is practically well. I have paid special attention to this point in my study of the disease, and hence feel justified in concluding that, while megaloblasts are a sign of serious import, indicating very far-reaching alterations of the blood-making organs, these alterations are not necessarily fatal.

Usually the leucocytes are diminished in number, but at times a leucocytosis occurs and is a favorable sign. Von Noorden has described a sort of blood crisis in certain cases, leucocytosis and a number of small red cells making their appearance simultaneously, this regeneration of red and white cells being always followed by a distinct improvement in symptoms. Leucocytosis does not speak, however, any more assuredly for a good prognosis than the presence of megaloblasts does for an unfavorable one.

Considering pernicious anæmia as an acquired anomaly of metabolism, it is not to be expected that its treatment would be very satisfactory. The prognosis is not, however, so bad as it used to be considered, so that there is more encouragement to take up seriously the therapeutics of the disease than there was when every case was considered to be fatally progressive.

First, of course, as in all diseases, we should remove the cause. If any of the factors that we have mentioned are at work, they must be eliminated. In order to get at causative factors, the most careful questioning as to minute details of every-day life, habits as to work, diet, etc., will at times be necessary.

To my mind, serious mistakes are often made at the beginning of anæmic conditions by telling patients, on general principles, that they must become red-cheeked once more by out-door exercise and sport, and that they must ride horseback or bicycle, or take long walks, perhaps in mountainous districts. This is often the very worst thing for them; what they need is rest, mental as well as physical.

What the poorer classes need above all is to be taken out of their surroundings and away from the work and the worry of home life. Hospital diet may not seem luxurious, but it is often for such people ever so much better than what they are accustomed to, and it is prepared properly and regularly served. For the better class of pa-

tients a change of scene is eminently suitable. It is, as a rule, the only way to secure that absolute mental and physical rest so necessary where overexertion seems to be the cause.

There are three main indications for treatment,—first, the loss of appetite; second, the auto-intoxication; and third, the constipation. They are all dependent on one another, so that improvement in one means also improvement in the others. The constipation is often the first link in the chain, and may be treated by salines and later by calomel occasionally, which acts as a purgative and an intestinal antiseptic. There should be daily lavage of the stomach and washing out of the large intestine, to get rid of toxines already present. As the auto-intoxication is overcome the appetite improves, except in so far as it depends on atrophy of the glands of the stomach and intestine.

As appetizers I prefer excitant food and piquant sauces to drugs; cheese, sardines, and pickles are often most satisfactory helps to an appetite for other things. The highly nutritious foods, such as shaved beef, scraped ham, with plenty of milk and thick soups and broths, should be given. Eggs should not, as a rule, be given in any amount, especially not in the finely divided state in which they are often put into soups. They are too liable to undergo decomposition, with the formation of the toxic albumoses, that it is our great effort to avoid. Hydrochloric acid and pepsin and the bitter tonics, especially nux vomica, are often of the greatest service as tonic appetizers. Certain bitter liqueurs, as Vermouth, are good. A favorite prescription of mine is a wineglassful of English porter before meals.

It is only after the general condition of the patient has been greatly improved that there should be any question of specific medication. Any drug, as a rule, will lessen the appetite, and that is particularly to be avoided. Iron, I think, is contraindicated, for this reason, in severe cases, and I have really never seen any good effect from it even later. There is already, as Quincke has shown, an abundance of iron in the system, but nature is unable to make use of it, and besides in the plenteous animal diet advised there is abundance of iron for the system's needs.

I have my patients weighed every eight days, and after they have gained in weight for three or four weeks I give the specific blood remedies, the principal of which is arsenic. We do not know how it acts; Professor Liebreich confesses that its effect on the organism is

a mystery, but it is of the greatest value. In order not to disturb the appetite, I give it always on a full stomach, and do not give it at the beginning of the treatment, when the desire for food is but slight, and it takes but little to lessen it. From iodine, which may be of use in old syphilitic cases, I have seen no good results. During two days of each week I substitute quinine for the arsenic, and think I have found it do good. The break in the administration of the arsenic prevents the system from becoming so accustomed to it as it otherwise would, and helps to put off the tendency to arsenical intoxication. From organotherapy I have seen no benefit. Bone marrow, fresh or compressed, does no good.

As rest and plenteous feeding constitute the principal part of the treatment, some have suggested the rest cure *régime* with massage. What the patients need, however, is perfect rest, and massage should be prescribed only when they are already well on their way to recovery. It is only when convalescence is well under way, too, that the question of patients going to the sea-shore or the mountains may be considered. Under such circumstances I have found the tonic influence and bracing air of the Baltic Sea or the Black Forest or Harz Mountains the best.

When improvement has come, it is important for the patient to remember that he is not cured. A defect of metabolism, liable to recur at any moment if any excess is committed, constitutes the essence of the disease. The diabetic and the obese know that they must ever be careful. In pernicious anæmia the same thing holds good. Seeming restoration to health will be rapidly followed by relapse, from which there may this time be no reaction, if the same causes are allowed to work once more. On the other hand, that the cure may with due care be lasting my own experience teaches me.

# TYPHOID FEVER COMPLICATING THE PUERPERIUM; THE DIAGNOSIS BETWEEN APPENDICITIS, TUBERCULAR PERITONITIS, AND TYPHOID FEVER; MENINGITIS; ABDOMINAL EFFUSION; PURPURA HÆMORRHAGICA; MEDIASTINAL TUMOR AND HÆMATURIA.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON MEDICAL COLLEGE HOSPITAL.

BY H. A. HARE, M.D.,

Professor of Therapeutics in the Jefferson Medical College and Physician to the Hospital, Philadelphia.

Gentlemen,—At the last clinic I showed you a large number of interesting cases, and I am glad to say that I have an equally interesting series to present to you to-day.

The first patient is a woman who was confined eight days ago and at the time of her delivery suffered from a post-partum hemorrhage. Immediately after delivery her temperature was found to be febrile, and since that time it has averaged from 102° to 103½° F. A careful examination of her pelvic organs and all other portions of her body fails to reveal any evidences of septic infection. The child has been taken from her and proper measures have been used to prevent the accumulation of milk in the breasts. To-day we find that she gives the positive Widal reaction, a few spots are appearing on her abdomen, her tongue is somewhat typhoidal in appearance, and from the character of her temperature curve there can be no doubt that typhoidal infection took place during the last two weeks of pregnancy, so that the disease manifested itself at the time of parturition. The case is a peculiarly interesting one, first, because it is generally supposed that pregnant women have a certain amount of immunity to typhoid infection; second, because typhoid infection complicating parturition opens up many possibilities which seriously increase the gravity of the case; and, thirdly, this patient is interesting be-

cause, notwithstanding her undoubted typhoid infection, her general condition is excellent, particularly in regard to the clearness of her mind and the absence of nervous symptoms.

The second case is also one of very great interest. This woman came under my care with the history that she had been ill for some days with chilly sensations, fever, and languor. The first twenty-four hours she was in the ward her temperature was in the neighborhood of 102° and 103° F., but within forty-eight hours after admission it fell to normal, where it remained for a week. She gave the history that for a number of months she had been suffering from constipation alternating with diarrhœa and abdominal pain. An examination of the abdomen reveals marked tenderness in the neighborhood of the appendix. There is also some thickening of the abdominal contents, so that about the umbilicus the intestines seem to be matted together. The abdominal wall is so thin that the knuckles of the intestines can be seen through it.

Under these circumstances, Dr. Keen and I agreed that she should be subjected to an operation, which should be exploratory in its character, and which would do good if it resulted in the removal of the diseased appendix or if it showed the presence of tubercular peritonitis. On the day on which she was to be operated upon, she complained of feeling very wretched, her temperature rapidly rose, and during the last four days has been distinctly febrile. Her tongue is coated, and this morning a copious rose rash appeared over her abdomen.

The question now arises as to the exact condition which was the cause of this woman's fever when she entered the house. Was the fever a primary attack of typhoid or the result of a chronic appendicitis or of peritoneal tuberculosis? Is the present attack of typhoid a relapse, or is it a primary attack superimposed upon appendicular or peritoneal trouble? Finally, what is the right treatment at the present moment, in view of her constant appendicular symptoms and the fact that she has distinct appendicular pain on palpation? I think that it is proper at the present moment to apply an ice-bag over the appendix and to treat her in every other way as though she had an ordinary case of typhoid fever. Should she at any time develop increased pain in the neighborhood of the appendix and rigidity of the abdominal wall, I think that an operation will be required, even if it has to be performed in the face of an attack of typhoid fever,

since her condition with the double infection would then necessarily be very grave.

I now wish to call your attention to the sputum which has been found in this spit-cup. It comes from a patient suffering from croupous pneumonia in the stage of resolution. You will notice that it still possesses the peculiar sticky quality, and it also contains a large quantity of muco-purulent material, which is a visible manifestation of the resolution process which, when we make a physical examination, we find is going on in the lungs. The stage of red hepatization is changing to the gray stage or that of resolution. She is getting, in addition to sedatives for excessive cough, carbonate of ammonium as a circulatory stimulant and chloride of ammonium as an expectorant.

The next case is one of tubercular meningitis occurring in a man of about twenty-five years, who presents a number of very interesting symptoms in general and some ocular ones in particular. You will remember that when I showed him to you three weeks ago he had excessive hyperæsthesia all over the body, particularly in the skin of the abdomen. The abdominal wall is very scaphoid, he is rapidly emaciating, and he has two small bed-sores developing over his sacrum. You will notice the peculiar vacuous expression of his face. His eyes are wide open and staring, the pupils are somewhat dilated, there is divergent squint, and you notice the constant mumbling which he carries on, varied now and again by a sharply spoken word uttered with the suddenness of a command. His eyes have been examined on two occasions by Professor de Schweinitz, the last time yesterday morning, and I will now read you the result of that examination,[1] which indicates that he is suffering either from hysteria or from meningitis; that the latter is the cause of his trouble

[1] Pupils semi-dilated; patient has been using "drops," probably a mydriatic; slow reaction to strong light; convergence reaction good; neither atrophy nor optic neuritis; veins a little fuller than usual; no extravasation in retina or chorioid; media normal; no palsy of external muscles.

Pupils show medium reaction, slow but normal; no failure in the ordinary rotation of eye, except outward to the right.

When fixing candle-light at one metre, there is $\frac{5}{100}$ of divergence (usually O. D.), and as one eye tries to fix the other wanders upward and outward.

At times spasmodic strabismus (sharp divergence of O. D.) may be demonstrated, also temporary left ptosis; there is no neuritis; some œdema around disk and full retinal veins.

The ocular signs indicate basilar meningitis.

we are almost certain, because his sex, the absence of other hysterical symptoms, the marked wasting, and the progressive character of his disease exclude hysteria.

You will remember that I spoke to you when he was last before us of the possibility of his having trichinosis. I have not as yet been able to get a piece of muscle to have it examined microscopically, but you will recall that recent studies of the blood in this disease have shown that there is an enormous increase in the eosinophiles. An examination of his blood shows that he has leucocytosis to a moderate extent, there being about fifteen thousand white cells to a cubic millimetre, but a differential count of these fails to reveal a single eosinophile. The possibility of the case being one of trichinosis is, therefore, still further removed. [An autopsy two weeks later revealed tubercular meningitis.]

The next patient is a woman of sixty years who, you will notice, has a very great enlargement of the abdomen. It is not enlarged in the broad and flat manner characteristic of ordinary ascites, but is pyriform in shape. Both flanks distinctly bulge, but the anterior abdominal wall is very much more dome-shaped than in ordinary ascites and the swelling is somewhat irregular in form. Over the lower part of the abdomen you will notice a peculiar deep-brown staining of the skin, which you sometimes see, usually to a less degree, in cases of grave abdominal disease. The woman tells us that about a year ago she noticed that her waistbands were a little too tight for her, and then suddenly six months ago she was seized with violent pains in the abdomen, with the feeling as if everything was being pushed up against her diaphragm, and then she noticed that the size of her abdomen was rapidly increasing. What are the possible conditions in her case?

The most common cause of accumulation of fluid in the belly—namely, cirrhosis of the liver—is to a certain extent excluded by her sex, by her history, and still more by the fact that there is no evidence of obstruction of the venous circulation in the abdomen. Only a very few small venules are to be found in the abdominal wall, and there is no venous engorgement nor the formation of the "caput Medusæ" so often seen in cases of ascites due to cirrhosis. The swelling is not due to any morbid growth, as, for example, a carcinoma of the liver, as it is very evidently due to liquid and is not a solid body. The abdomen has not the shape nor does palpation re-

veal the signs usually met with in peritoneal tuberculosis. We must also consider the possibility of its being an enormous ovarian cyst, ruptured or unruptured, or a ruptured cyst of the broad ligament. The history that there was sudden increase in the size of her abdomen would seem to point to the probability of its being a ruptured ovarian cyst, the fluid having continued to be poured out since the rupture took place; or, again, we must not forget that it is possible that this is an ovarian cyst with a twisted pedicle, which would account for the violent pain from which she has suffered. Because of the obscurity of the case and because of the distress which she suffers by reason of the fluid, I am going to determine by aspiration its character, and if the results are favorable will draw off a considerable quantity of it. I shall also test the fluid that I obtain, because it may throw some light on her case. You remember that in abdominal effusion due to dropsy the liquid usually contains a trace of albumen, about 0.97 per cent., and has a specific gravity of from 1.006 to 1.008. The same characteristics hold good concerning the fluid obtained in cases of cirrhosis. On the other hand, diseases of the peritoneum usually cause the formation of a fluid having a specific gravity of 1.018 to 1.027, and a comparatively large quantity of albumen, varying from 3.8 to 7.10 per cent. The fluid of an ovarian cyst usually has a specific gravity of about 1.026, while that of the broad ligament usually has a much lower specific gravity. There is still another test which I am about to make in this case. In cases of ordinary transudation into the abdominal cavity we can frequently obtain iodine in the fluid if it is administered a short time before the tapping is done, its presence being determined by adding to the fluid some chloroform and some nitric acid, when the characteristic color of iodine is developed. I gave to her this morning ten grains of iodide of sodium. If the liquid is the result of some chronic disease of the peritoneum iodine will not appear for a long time, if at all; if, on the other hand, it is a transudation from the blood-vessels it will appear quite promptly.

I now make the puncture, and on using the syringe I withdraw a quantity of dark-red, somewhat grumous-looking fluid. When subjected to the test that I have mentioned it fails to show iodine, but you will notice that it is very largely albuminous, forming a tremendous precipitate on the exposure to either heat or nitric acid. Because of the bloody appearance of the fluid, I am inclined to believe

that the patient has either a malignant growth or else an ovarian cyst with a twisted pedicle, and I shall ask Dr. Montgomery, our professor of gynæcology, to see her with me in consultation.

The next case is one of purpura hæmorrhagica occurring in a man of thirty-five, a tailor by occupation. I have not time to speak about this curious condition, save to point out to you that he has had rheumatic manifestations and that purpura is supposed to be frequently associated with rheumatic affections.

The next case is in a man of sixty-five, who complains of shortness of breath and a sense of discomfort in his larynx. An examination of his chest is negative so far as his lungs and heart are concerned, but he has a distinct area of dulness half as big as my hand to the left of the sternum, extending from his first rib to the third interspace and almost to the anterior mammary line. A laryngoscopic examination reveals that his right vocal cord is in the correct position for phonation, but his left vocal cord is in the cadaveric position, indicating that he has paralysis of his left adductors. If he had bilateral paralysis he would be speechless, but he now phonates chiefly with his right cord. He, therefore, probably has pressure, by reason of some mediastinal growth, upon his left recurrent laryngeal nerve. The most probable cause of his difficulty is an aneurism of his aorta, but there are none of the physical signs of such a lesion save the dulness on percussion, either anteriorly or posteriorly. He has no unilateral sweating of the face, no dilatation of the pupil, no hyperæmia of the vessels of the face or neck. If he has not an aneurism he may have a sarcoma of the glandular tissues in his mediastinum; but this is unlikely, because of the absence of any signs of cachexia and of the moderate symptoms which he presents. If he has not a sarcoma he may have a mediastinal abscess, but this is also set aside by the absence of any of the symptoms usually associated with the presence of pus. The other forms of mediastinal growth are almost excluded by their rarity, and, notwithstanding the absence of physical signs, I am inclined to think that he has an aneurism of the descending aorta so situated that it does not produce external manifestations. I forgot to tell you that he has difficulty in swallowing.

The last patient is a man of forty-five, who came to us three weeks ago suffering from marked hæmaturia which has persisted ever since. He states that he feels perfectly well save that he is losing

slightly in strength, probably because of the loss of blood. You will notice the peculiar bloody appearance of the urine. A microscopic examination of this reveals red blood-cells, little altered in appearance and not crenated; his urine is alkaline, and probably the cells have not been floating free in the urine for any length of time. That he is not suffering from acute nephritis seems probable by the lack of symptoms of this condition, and by the absence of any vertigo, headache, or other sign of uræmia. That it is not a renal calculus eroding a vessel is shown by the absence of any history of pain, which you remember was a prominent symptom in the patient that I showed you and upon whom Dr. Keen operated and found a renal calculus. That it is not hemorrhagic infarction of the kidney is shown by the absence of all cardiac and circulatory lesions. Malarial hæmaturia is also excluded, as is also the possibility of its being a toxic nephritis, by the symptoms and by the lack of any history of his having swallowed irritating poisons. There is also no history of any injury which could have resulted in damage to the kidney. These things being excluded, we must consider the possibility of his having a papilloma of the bladder or some other lesion of this viscus, and to decide this point positively we will ask the professor of genito-urinary surgery, Dr. Horwitz, to examine him and let us have a report as to his vesical condition.

# HYSTERIA AND RAYNAUD'S DISEASE.[1]

CLINICAL LECTURE DELIVERED AT THE FRANZ JOSEPH HOSPITAL.

BY P. KOVACS, M.D.,

Clinical Lecturer of Franz Joseph Hospital, Berlin, Germany.

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GENTLEMEN,—We have here to-day a young lady, twenty-one years of age, whose principal occupation is washing dishes for restaurants. She complains of nervousness and a discoloration of her fingers.

The history she gives is that for the last four months she has noticed that the moment she put her hands in water they became pale, and, at the same time, she experienced a peculiar tingling, burning, and sticky sort of pain in the tips of her fingers. As soon as she took her hands out of the water they commenced gradually to return to their normal color. This phenomena continued until four weeks ago, when, instead of gradually returning to normal, the pale color would assume a bluish hue, and, if she went indoors or into a warm place, the color would change from a blue to a red, later returning to normal. This play of colors was first noticed on the index finger of the right hand, and it gradually spread to the other fingers and thumbs of both hands. During the last two weeks the color has become a permanent blue, and it is for this she comes to us to-day. At the present time when she washes her hands in cold water her fingers turn yellow, and the remainder of the hands, together with the wrists, turn blue. In the beginning of the trouble, when the color used to ultimately return to normal, she noticed a loss of sensation in her fingers, and also observed later that even if she merely went out in the cold air they would turn blue.

We will now proceed with the examination of the case. Upon looking at her, her general appearance contraindicates any chronic or wasting diseases, and you will also notice that she has a peculiar

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[1] Reported by H. Artelt, M.D., Philadelphia.

nervous appearance and manner, answering in a quick, snappy, but nevertheless a distinct, manner.

The examination of the heart and lungs is negative, except that there is a systolic murmur heard over the entire body of the heart, loudest over the pulmonary area. There is no accentuation of the aortic second sound, and examination of the temporal, axillary, brachial, and radial arteries shows that they fill regularly, and that they are soft and easily compressible.

She tells us that she has noticed that the left half of her body is weaker than the right half, but we only get this information after we have made our tests of her strength by comparing both sides.

We will now proceed to a thorough examination of the patient.

You will observe that the head is mesocephalic.

The vertebral column shows no scoliosis, pressure symptoms, nor tender spots, and the head likewise is negative as to the latter symptoms.

Cerebral symptoms or signs are absent, but she has occipital hemicrania.

The twelve cranial nerves give negative results, and there are no transitory nerve changes when the attacks come on.

Abdominal muscles negative.

Lower extremities negative, except for the diminished power of her left limb, mentioned above.

*Upper Extremities.*—You will notice that her fingers are cyanotic, which reaches up to the third phalanges, and become deeper in color as we go towards the tips of the fingers. By looking carefully

at the tips we notice that three or four of them show small cella or cuplike excavations; also, on examination of the nails, we observe small, black, striped spots which, on close examination, are found to be hemorrhagic in character.

The hands are cold and clammy on the palmar surface, and the

skin over the last phalanges is hard and sclerotic to the touch. There is no muscular atrophy or fibrillary contraction.

There is no incoördination, but on the finer movements of the fingers the right hand is impaired.

Periosteal and biceps flexis are normal.

*Sensibility of the Hands.*—She has no feeling of anything wrong, although at first, when the fingers became cyanotic, she had the peculiar tingling, burning, and sticky sort of pain, already mentioned, in the right hand. Later the left also was affected, but since the fingers became permanently cyanotic, she has only had periodical pains, and at the present time complains merely of a feeling of numbness of both hands.

As to the temperature changes of the hands at the present, if she puts them in cold water they turn white, later assuming a blue color; but if she puts them into hot water they turn red, later turning blue.

| RIGHT SIDE. | LEFT SIDE. |
|---|---|
| *Finer Sensibility.* | *Finer Sensibility.* |
| Decreases as it descends from the upper part of the forearm until, when it reaches the hand, it is completely lost. | Decreases as it descends, but the thumb has lost it completely and the fingers only from the second joint down. |
| *Rough or Deep Test.* | *Rough or Deep Test.* |
| There is complete anæsthesia of the end of the thumb and all four fingers up to the metacarpo-phalangeal joint, together with the skin over the joint. | Not lost completely at any place, but greatly decreased all over. |
| *Pain.* | *Pain.* |
| Needle pricks are called "slight pressures" from the elbow down to the hands, and the fingers show complete loss of pain. | From the hand down the sense of pain is greatly decreased. |
| *Temperature.* | *Temperature.* |
| Extreme changes cannot be recognized from the elbow down. | The same as on the right side, except that it starts from two inches to three inches below the elbow. |
| *Steriognosa.*—(Testing the sensibility by placing objects in patient's hands for diagnosis, as key, spoon, etc.) | |
| Shows sensibility to be greatly decreased, the patient rarely being able to name the object. | |

As a result of these tests, what do we find?

1st. That there are motor disturbances.

2d. That there are sensory disturbances.

3d. That there are tactile disturbances.

4th. That there is slight hemiparesis.

5th. That there is hemihipanæsthesia.

6th. That there is no loss of coördination.

7th. That there are no spastic symptoms.

8th. That there are no bladder or rectal symptoms.

So that, taking all in all, it points strongly towards hysteria. But have you been observing close enough to see that this hand trouble has presented three stages or stadia, the first of which, one of local anæmia, was most likely due to local vasomotor disturbances, and being periodical excludes all diseases but one, which will be mentioned later? That it was a spasm of the vessels was clear, and is known as the stage of local syncope. It then went into the second stadium, or cyanotic stage, which is due to a narrowing of the veins by spasms, and is called the stage of local asphyxia. From this it went into the third stadium, or stage of vessel paralysis or gangrenous stage, in which she had the red coloration of the fingers with a sensation of heat and loss of substance, later represented by the cella or cup-like excavations on the tips of some of her fingers, already mentioned and observed.

These three stages belong to vessel neuroses, and the disease is often known or spoken of by the laity as the washerwoman's disease.

The symmetrical appearance of the disease, the fact that there is no muscular atrophy, and that it presented three stages, all point towards Raynaud's disease.

Another disease that somewhat resembles this is angioneurotic œdema, in which we have a sudden swelling of some part with œdema, the œdema lasting only for a few hours and giving rise to no symptoms. It is, however, important to remember this condition, as it may attack the glottis, causing death by suffocation.

Another disease is syringomyelia. But in this affection the vasomotor disturbances are not so marked, and another symptom, sclerosis, which is absent here, would have to be present; also, in syringomyelia, you would by this time have marked muscular atrophy.

We can therefore, with perfect safety, say that this is either a

case of hysteria with Raynaud's disease, or Raynaud's disease with hysteria.

The line of treatment was principally electricity and massage, together with cotton gloves to keep a constant warmth in her hands, with a soft diet and tonics. The patient was seen every other day, and the above treatment almost immediately gave good results. In about three weeks' time the patient was brought before the class again, merely as a matter of interest, both hands having entirely returned to their normal appearance. She was discharged with the advice that, if possible, she should change her vocation and engage in an occupation in which she was not required to do most of her work in water.

# REPORT OF A CASE OF RIGHT SUBCLAVIAN ANEURISM.

BY HENRY L. ELSNER, M.D.,

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This case is brought to notice because in it will be found all of the classical symptoms of subclavian aneurism. Indeed, the symptoms and physical signs are so characteristic that they correspond exactly with the description of the disease as we find it in our leading text-books and monographs on the subject.

The patient is a fruit dealer, sixty-six years of age, with a negative family history. He denies specific disease, but admits that during the greater part of his life he has indulged freely in the use of spirits and malt liquors. He has used tobacco, but not inordinately. He denies other excesses. During the later years of his life he has daily visited the offices in some of the higher buildings of the city for the purpose of selling fruit, and was therefore obliged to climb stairs. The effort has of late become less easy than formerly, owing to transitory dyspnœa from which he has occasionally suffered. He never had any serious illness, but has been unusually well. Until he first noticed the symptoms of his present illness, his muscular development was good, and his digestive and other functions were normally performed.

In December of last year he suffered from what he supposed was a slight cold, but noticed that, as the catarrhal symptoms disappeared, he had more or less shortness of breath, which he described as located in the upper part of his chest. To use his own words, he "was caught for breath up very high." Neither at this nor at any other time since the beginning of the disease has there been pain. The dyspnœa became more and more distressing from day to day, until at last he was forced to give up his daily canvassing and confined himself to the sale of fruit at home. While he says that he had no acute pain, he

complains at times of a peculiar pressing sensation in the upper thoracic regions of both sides, but most annoying in the right infraclavicular and mammary regions.

About the middle of January he noticed what he describes as a beating in his "*right throat.*" He neither complains of numbness of the right arm, swelling, nor coldness; there is no dysphagia, dyspnœa, vertigo, tracheal tugging, hoarseness, nor cough. He complains of constipation, which has become more obstinate since the beginning of his present sickness. He says that his bowels have not moved naturally during the past six weeks; for relief he has been forced to use salines daily. The pupils are moderately contracted, react normally to light and accommodation, and are not unequal.

Physical examination shows dilatation and prominence of all the veins covering the right half of the thorax and extending down the right arm. There is no œdema of the arm, neither is its temperature as compared with that of the left perceptibly changed. The patient, while he has grown thin, has not become anæmic, in spite of the fact that his appetite is not as good as it formerly was. Inspection still further shows the supraclavicular, the clavicular, and the infraclavicular regions abnormally prominent in their inner and middle thirds, particularly the first two regions mentioned.

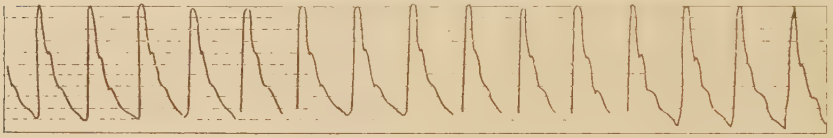
In the supraclavicular region there is an abnormal prominence, which presents a pulsating tumor, and which on palpation is found to be continuous with the right subclavian, gives fluctuation, and is very thin, as you can easily recognize by palpating, at its most prominent point. Palpating the mass laterally, we find characteristic lateral expansile pulsation. The entire mass is about the size of a large goose-egg. The clavicle and the first and second ribs near the sternal articulation are pushed forward, and in the first right intercostal space deep pressure elicits pulsation. Over the prominent portions of the regions mentioned the percussion note is abnormally high-pitched and dull; over the pulsating mass there is absolute flatness.

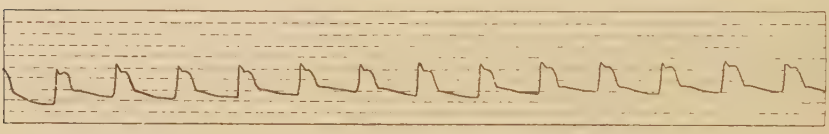
Auscultation over these regions, particularly over the tumor, discloses a systolic bruit, and this is easily recognized also when the sides of the mass are ausculted. The apex beat is found slightly to the left of the mammary line, in the fifth interspace, the diffuse cardiac impulse is unusually weak, and the area of cardiac dulness is increased slightly to the left and to the right of the normal cardiac line. On auscultation the heart-sounds are found distant, embryonic, and un-

usually feeble. In the aortic region there is no increased lateral area of dulness; the first sound is muffled and the second sound accentuated, with an associated metallic click.

The pulse at the time of first examination was normal in frequency; but it was evident that the right radial was less full than the left, and that the systolic contraction was associated with less force on this side than on the left. The accompanying sphygmographic tracings show very prettily the inequality in the two radial pulses. It would seem as if the blood flow from the heart through the right radial was in an almost continuous stream, and at times, to the finger at least, the rise and fall of the pulse wave were almost unnoticed.

The urine is acid, contains neither albumen nor sugar, and its specific gravity is 1.027. Microscopic examination is negative.





With this history there is no doubt of the presence in this case of an aneurism involving the right subclavian artery. The interesting features are the associated dilatation and enfeebled right and left hearts, and the absence of compensatory change in the left ventricle, which so often precedes or is associated with aortic, thoracic, and other aneurisms involving the peripheral arteries.

Another interesting feature is the absence of evidence of arteriosclerosis in any of the superficial or palpable arteries. The temporals are not particularly prominent, though at times of undue excitement there is a very slight tendency towards tortuosity.

Still another interesting feature has been the absence of pressure symptoms at any time during the course of the disease, though there has been sufficient force to elevate the clavicle and the ribs; none of the functions of the trachea, larynx, or œsophagus have suffered.

The thinness of the aneurismal wall without evidence of coagulation of blood to protect the artery is rather ominous, and it has seemed to the surgeon, as well as to myself, that the positive evidences of myocardial degeneration with dilatation and lack of compensation, associated with a very thin sac wall, preclude the possibility of doing anything for this man surgically.

It would be rather hazardous, with an artery the coats of which are almost ready to rupture, to use any of the methods by which coagulation is usually favored, either by the introduction of a wire, needle, or any one of the other foreign substances which have been recommended in the past, and occasionally successfully used if recent clinical reports can be trusted as giving sufficient data to warrant conclusions.

The treatment of the patient has seemed, in a degree at least, to relieve the dyspnœa, and he believes himself to be freer from annoying symptoms than he was before it was instituted. Besides absolute rest and the old-fashioned Tufnell diet, the patient has received, since the 26th day of April, increasing doses of a saturated solution of the sodium iodide.

In connection with this case I recall a patient in whom I diagnosed aortic aneurism from the presence of tracheal tugging, aphonia, and increased lateral dulness in the aortic area, at one of my clinics recently. The diagnosis was ultimately confirmed by post-mortem examination. The patient died suddenly from the rupture into the trachea of an aneurism of the arch of the aorta.

A number of years ago I had a very interesting case of subclavian aneurism, also of the right artery, in which the patient finally died from rupture of the heart. In this case, four or five days before death, there were sudden syncope, pallor, thready pulse, and evidences of collapse, from which the patient recovered and in the course of a few days died suddenly. At the post-mortem, besides the subclavian aneurism, I found the heart muscle degenerated, with a partial rupture and hemorrhage into the myocardium, involving the left ventricle, and a rupture of the left ventricle below the aorta. Undoubtedly the partial rupture caused the symptoms which preceded the sudden death by a few days.

# THE ROENTGEN RAY DIAGNOSIS OF RENAL AND VESICAL CALCULOUS CONDITIONS.

BY CHARLES LESTER LEONARD, A.M., M.D.,

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The difficulties that surround the differential diagnosis of suppurative conditions in and about the genito-urinary tract are very great. There are, of course, many cases in which the symptoms are so clearly defined that the diagnosis is practically made. Even in these cases, however, there is always an element of indefiniteness that makes greater precision desirable.

In cases of vesical calculus the diagnosis can generally be made from the classical symptoms which usually render it unmistakable, so that confirmation by the vesical sound seems almost superfluous. Yet cases occur where these symptoms are entirely absent, and where it is impossible to detect the calculus even with the sound in the bladder.

In a recent paper in the *British Medical Journal*, Mr. W. Bruce Clarke says, of encysted vesical calculi, "The ordinary symptoms that we are accustomed to associate with vesical calculus are frequently absent in such cases, and consequently the diseased condition assumes considerable proportions before it is detected. A stone which is fixed does not, as a rule, give rise to hæmaturia, and the acute pain, which is generally a consequence of the stone becoming grasped by the neck of the bladder, is usually remarkable by its absence. It may further be added that in some cases the stones are so deeply embedded in the bladder wall as to elude the beak of the sound, and only proclaim their existence when an attack of cystitis has supervened."

He says, in addition, "It is difficult to single out any one sign which might be held to be of diagnostic value." "The difficulties of the diagnosis were enhanced by the fact that in several instances

washing out the bladder was quite sufficient temporarily to restore the urine to a healthy condition. It is perhaps also worthy of note that in several instances the stones when extracted proved to be so thickly coated with mucus that even when they lay upon the table no audible note could be obtained when they were struck with the beak of the sound. This affords a ready explanation of the difficulty which may be experienced in detecting a stone in the bladder under certain conditions."

There are many cases of enlarged prostate that are complicated by the presence of vesical calculi; cases that, after the calculi are removed, so rapidly improve in their condition that further operative interference is unnecessary. It is, however, next to impossible to detect these calculi; they lie behind the prostate in a deep pouch of the atonic bladder, far from the reach of any exploring sound that can be passed beyond the prostate.

In more than half the cases reported by the author quoted above, the presence of stone was not suspected before the operation. So impossible is the diagnosis that he advises suprapubic cystotomy as a diagnostic measure in cases of chronic cystitis that remain rebellious to treatment.

The differential diagnosis in calculous disease of the kidney is even more difficult, since the conditions that may simulate it in symptomatology are much more numerous. Even where the classical symptoms are present and the diagnosis is certain, as in the simple cases of vesical calculus, there is an indefiniteness or lack of detail that is a manifest disadvantage. But the classical symptoms of calculous nephritis are frequently absent, or, at least, the symptom complex is such that differentiation is impossible, and, even where the symptoms have indicated the presence of a calculus, operation has failed to detect it, possibly because the calculus was encysted and the diagnosis lacked the detail of its exact location, possibly because the symptoms due to some other disease had so closely simulated it as to make the differentiation impossible.

The cases in which the diagnosis is most evident are those in which the patient has passed calculi on former occasions and where there have been frequent attacks of renal colic, with a persisting tenderness and pain in the lumbar region that is increased by violent exercise or jarring. The urinary analysis shows calcium oxalate or uric acid, with an intermingling of blood that varies in quantity.

A review of those renal conditions, the symptoms of which approach so nearly those of calculous nephritis as to render a differential diagnosis necessary, will show how difficult the diagnosis often is, especially as one or more symptoms are frequently absent.

The different varieties of pyelitis and pyelonephritis have a group of variable symptoms that can be very readily confused with calculous nephritis; in fact, they differ frequently from nephrolithiasis only in the absence of the calculus. The hemorrhagic forms show the presence of both white and red blood-corpuscles in the urine, intermingled at times with pus, while there are in the loin pain and tenderness that are referred to one kidney. The severer forms of pyelonephritis and pyonephritis have much the same symptomatology, although the more acute symptoms of pain and tenderness in the region of the kidney are absent and the urine is generally free from red and white blood-cells. In many of these cases the kidney has been opened and searched for calculi, in the belief that they were true cases of calculous disease.

The tumors that develop in the kidney frequently give rise to symptoms that make the differentiation difficult. In the early stages of malignant or adenomatous disease we have pain referred to the kidney and a variable amount of hæmaturia that is not unlike that said to be characteristic of calculous disease.

In tuberculous nephritis there is a condition in which calculi may form in conjunction with the disease and in which it is always difficult to prove the true nature of the infection. The microscopic examination often fails to reveal the presence of the tubercle bacilli; blood, pus, epithelium, and fragments of tissue are common to both conditions. The most certain test is the injection of the pus-containing urine into a guinea-pig and subsequent study of the changes that follow. But even this test may fail. The tubercular changes may not take place or the tubercle bacillus may not have been present in the urine employed.

In the varying degrees of mobility found in movable and floating kidneys we find the exciting cause of many symptoms that render the differential diagnosis of these conditions from calculus very difficult. Slight mobility, that cannot be detected by palpation, may give rise to congestion by twisting or sharply bending the renal vein, and produce intermittent hemorrhage. Or the sharp flexion of the ureter may so retard the flow of urine as to cause a hydronephrosis

that is intermittent in character, and sets up a pyelonephritis that will simulate calculous disease so closely as to defy the best diagnosticians.

The inflammatory changes that supervene after the passage of a small calculus through the ureter or the condition that arises after an acute inflammatory process in that part of the genito-urinary tract are not so clearly defined as to make their differentiation from encysted calculus very easy. The numerous cases reported demonstrate the difficulties that lie in the way of exact diagnosis in the varying inflammatory conditions which we find in the kidney. They also testify to the inability of the most expert diagnosticians to form an absolute diagnosis, and show that those most conversant with the diseases of the genito-urinary system realize the impossibility of rendering an absolute diagnosis in many of these cases.

The difficulties in diagnosis are not, however, confined to those diseases of the kidney that simulate nephrolithiasis. There are many conditions in diseased organs around the kidney that may produce symptoms so similar to those of renal calculus that the diagnosis is impossible. The proximity of the appendix may make a diagnosis difficult. I have seen a chronically inflamed appendix and the acute attacks which had from time to time taken place so simulate the impaction of a calculus in the ureter as to render the diagnosis very difficult. In the female we have in addition the diseases that involve the ovaries. Here the difficulty is increased by the fact that in true calculous disease the sensation of pain is frequently referred to the ovaries.

Seminal vesiculitis is another inflammatory condition of the genito-urinary system that, although it is readily detected by careful examination, has a symptomatology which is so varied in its manifestations, with pains occasionally radiating into the testicle and loin and often accompanied by cystitis, that it may be confused with the late stages of calculous disease in the kidney or bladder.

The literature of this subject is filled with reports of cases where operative interference for suspected calculi has only proved that one or other of these conditions had so simulated calculous disease as to be misleading.

If the cases that simulate calculous disease were the only ones in which a differential diagnosis had to be made, the task would be difficult enough. We know, however, by clinical experience and post-mortem examinations, that calculi may exist for a long time in

the kidney and give rise to no marked symptoms, and that the process of destruction may progress to such a degree as to destroy the integrity of the kidney and yet remain unsuspected. These unsuspected calculi should be found. There must be at some time in the history of these cases sufficient symptoms present to make a careful clinician suspect their presence. Like their fellows, the quiescent calculi, they are a constant menace to the individual. The presence of a calculus in a kidney, no matter what its size, is a very grave menace to the integrity of that organ. What if some become encysted? What if some are passed or become quiescent? Have we the right to let our patients take the chances of impaction and of calculous anuria because a calculus that is present is not producing acute symptoms?

Whenever a calculus is present anuria may at any time follow its impaction. No more deadly condition is known than complete anuria. It develops insidiously; till near the close there are no symptoms present other than the suppression of urine. Pain is not a constant symptom, nor is it always located at the seat of the obstruction. Have we the right to tell our patients that because these symptoms have ceased there is nothing to be feared, nothing to be done?

Statistics have shown that early operation or nephrolithotomy is attended by a very low mortality, while nephrotomy or later operation and nephrectomy are much more serious, with an average mortality of at least twenty-five per cent. Early operation is, therefore, of great advantage to the patient. How do we know that a calculus that has not been seen to pass *per urethram* has left the system? It may have become impacted in the ureter lower down. The numberless cases of calculi encysted in the ureter, that have been found clinically or in post-mortem examinations, makes this highly probable. The calculus may have completely occluded the ureter and by backing up the urine have totally destroyed the function of the kidney. How can any one say positively that medicinal treatment of any sort has freed a patient from a calculus and the dangers that threaten from its impaction? The relief of symptoms may be altogether due to the impaction of the calculus and the functional destruction of the kidney. The presence of such an impacted calculus in one ureter, followed by the destruction of the kidney on that side, places the patient in imminent danger of anuria, should calculous conditions be present or arise in the other kidney. A "cure" by medicinal measures is but the establishment of a false sense of security, while those cases

that have been cured by internal medication are, more than likely, those that were not really calculous disease, but in which the symptoms closely simulated it.

Exploratory nephrotomy was the best method of treatment in these cases. The practitioner had no right to allow his patient to run the risk of complete anuria because he believed that the cessation of symptoms meant a cure. Exploratory nephrotomy and nephrolithotomy are not dangerous operations. Their mortality in competent hands is less than three per cent. No patient should be allowed to risk the loss of one kidney or the danger of complete anuria because his symptoms have temporarily ceased.

Calculous nephritis is due to the presence in the system of an excessive amount of one of the chemical constituents of the urine that pass in a too concentrated form through the kidneys. For this reason we are liable to find calculi in both kidneys. Clinical experience and post-mortem examination have shown that this is not an infrequent occurrence. Operations in such cases have clearly demonstrated its great danger, but the only suggestion for its relief has been double exploratory nephrotomy.

This brief review of the symptomatology of renal and vesical disease and those conditions that simulate calculous disease in their development is sufficient to point out the difficulties that present themselves in the establishment of an absolute diagnosis. In many instances an absolute diagnosis is impossible and in the great majority early diagnosis is absolutely precluded, yet early diagnosis in calculous disease is essential to a low mortality in operating and to the preservation of the function of the kidney, while the presence of small undetected or quiescent calculi is a source of grave danger from impaction and anuria.

The Roentgen-ray method of diagnosis offers a solution for all the difficulties that surround calculous disease of the kidney and bladder. By this method these foreign bodies, formed within the system, may be detected and their size and exact location determined with mathematical accuracy. Not only can all calculi which are present be found, but we can in other cases say with absolute certainty that there are no calculi in the system. This absolute negative as well as positive diagnosis has been accomplished by developments in technique and apparatus whereby differentiation in the lesser densities of the softer structures can be secured. Thus the outline of the

kidney itself or of the erector spinæ muscles in the lumbar region can be shown in the negatives. Where such detail in tissues less dense than the least opaque calculus can be shown, we can be certain that no calculus is overlooked.

The difficulties in the technique of this diagnosis are not as great as might be supposed, now that an improved self-regulating tube has placed in our power the production of the varying qualities of Roentgen discharge in sufficient volume to penetrate any portion of the body.

The light employed in the diagnosis of calculous conditions is produced in a tube having a vacuum the equivalent in resistance of one to two inches of spark in air as measured by the parallel spark-gap on the induction coil. What, however, is essential is that the secondary current shall have enough ampèrage to produce a volume of Roentgen discharge from the low-vacuum tube employed. In other words, the efficiency of the spark of an induction coil or of any spark that energizes an x-ray tube, is to be judged by its measurement in watts and not by the number of inches of air resistance it will overcome. The coil employed in the examination of the series of cases upon which this paper is based has but an eight-inch spark in air; in watts it is, however, equivalent to many fifteen-inch coils.

The distance of the platinum of the anode from the plate is about fifteen inches. The patient is always protected from "burns," the effect of the electricity surrounding the tube upon tissue, by the interposition of a grounded aluminum or gold-leaf screen, which is placed between the tube and the body.

The chief difficulty is found in adapting the length of exposure to the individual case and in being certain that the tube is giving out the requisite volume of x-ray discharge. The exposure varies in length from five to fifteen minutes, according to the individual case, and it is sometimes necessary to make three or four exposures before a series of plates that have sufficient detail are produced. More than one plate is essential, to prevent errors that might arise in faulty coating of the plates. When, however, two or more plates are obtained that show a calculus distinctly, no matter how minute it is, the positiveness of the diagnosis cannot be questioned.

The series of cases in which this method of diagnosis has been employed furnishes an illustration of the difficulties to be encountered. Some were sent because it was impossible to establish a diag-

nosis and to exclude calculus. Of the thirty-eight cases examined, only twelve had sufficiently marked symptoms to justify operation. In seven of them calculi were found, while the remainder confirmed the accuracy of the negative diagnosis.

The following were shown to be the advantages of this method:

An absolute negative as well as a positive diagnosis can be made in every case.

The patient is saved the danger, inconvenience, and suffering consequent upon an exploratory operation, and all operation is avoided except in cases where it offers an absolute cure and freedom from all pain.

The details afforded regarding the number, size, and position of the calculi, and the solution of the question of the involvement of the other kidney, add an element of mathematical accuracy to the most positive diagnosis that can be made by other methods, and render the operation absolutely complete, as no calculus can escape.

In cases of anuria this mathematical accuracy is invaluable, and makes operation intelligent, where the symptoms present are at least vague.

The early period at which an absolute diagnosis can be secured saves the patient from the dangers of impaction and the destruction of the kidney, while furnishing indications for immediate operation at a time when statistics prove that there is the least danger.

In cases of encysted vesical calculi exploratory suprapubic cystotomy is avoided, and yet where calculi exist they can be found and the cure of a troublesome condition secured.

# THE COLD WAVE OF FEBRUARY, 1899.

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While great interest attaches to the areas of low barometric pressure as they pursue their course across our continent, the areas of high pressure are not without their interest and importance. In the area of low pressure resides the cyclone, while the area of high pressure determines the anticyclone. A cyclone is an entirely different thing from a tornado. It is a very broad storm, oftentimes a thousand miles in diameter, and sometimes can be followed half around the world. The winds of the cyclone are at a distance from its centre, and occasionally rise to the violence of a hurricane, but they are not to be compared to the extreme violence of the tornado, as, for instance, the recent one in Kirksville, Missouri, before which the most solid structures were razed. The cyclone is accompanied by clouds and rain; the anticyclone is cloudless, bright, and invigorating. However much more to be desired the anticyclone may be than its opposite, there are yet some things to be feared from even its advent. I refer to the great cold waves that in winter sweep across the continent.

We all recall that after some especially severe storm in winter the sky is clear, the wind dies away, and on the following morning the still, sharp air, the azure sky, and the creaking of wheels proclaim that the mercury is near the zero mark. That is the winter anticyclone. Stillness instead of the whirlwind; dryness instead of moisture; intense cold instead of a temperature of 30° or 40° F. These cold waves are most intense when an area of low pressure has passed to the northeastern corner of the United States, and when, perhaps, another low area is developing in the southwest, the high area being situated in the northwest. If, now, isothermal lines are

crowded together, owing to the close proximity of warm air to the colder air of the northwest, we expect a sudden and severe fall of temperature over a large extent of country. To constitute a cold wave, so called, there must be a fall of twenty degrees or more in twenty-four hours, free of diurnal range, and extending over an area of at least fifty thousand square miles of country, the temperature somewhere in the area going at least as low as 36°.

By drawing lines on a map through the places where an equal fall of temperature has occurred certain areas will be enclosed, and these are occasionally of very large extent. In one of the greatest cold waves of recent years, that of February 17, 1883, the temperature at 7 A.M. was twenty degrees lower than at the same hour on the day preceding throughout an extent of over one million square miles, extending from Lake Superior and Georgian Bay on the north to the Rio Grande on the south, and from Kansas City to Cincinnati. Inside the area of twenty degrees fall there was an area of thirty degrees fall of six hundred and forty thousand square miles; inside of this an area of forty degrees fall of one hundred and eighty-seven thousand square miles; inside the forty degrees fall was an area of thirty-one thousand square miles of fifty degrees fall, and inside of this a sixty-degree fall at Keokuk, Iowa, the centre of the cold wave, the temperature of that place being 60° F. on the morning of February 16, and zero the next day.

In the winter these cold waves are frequent and, unless exceptional, do comparatively little harm. We expect them to occur, and usually feel the benefit of the invigorating air. But as spring opens and vegetation becomes active, as people are inclined to throw off their clothing of winter, there is a sudden arrest of the warm weather; the sun's warmth, which seemed to favor the growth of crops, which inspired hope in the breasts of thousands of invalids, meets with a rebuff. The season is reversed, the sun's rays are weak, and every one is anxious to know the effect of the cold snap on their various interests.

It seems fitting that we should make a note, at least, of the remarkable period of cold which prevailed over the greater portion of the United States during the second week in February of this year. It was an unparalleled cold wave, and established a new record for the majority of stations east of the Rocky Mountains. Three cold waves covered the period from January 26 to February 14, and fol-

lowed extensive snow-storms which caused great distress and loss of life in the Western States. A detailed study of the atmospheric conditions has been made by Mr. A. J. Henry, of the United States Weather Bureau, and published in the monthly *Weather Review* for April. It may be said, in brief, that the snow-falls extended as far south as Northern Texas late in January. The Rocky Mountains seemed to act as an effective barrier to the movement of cold air westward, so that after February 7 the condition to be mentioned seemed to have only a moderate influence on the Pacific slope. On February 1 an area of low pressure moved inland from the Pacific, striking the continent about latitude 45°, and proceeded across the Great Basin towards Southern New Mexico and Southwestern Texas. This low area passing into the interior made it possible, therefore, for the rapid descent of cold air from the areas of high pressure. Just as water flows down hill, so cold air flows from high to low, and the more rapid the sharper the gradient. This was, then, the keynote of all the changes that followed. This low pressure was accompanied by heavy snow-storms.

The second high-pressure area started from Assiniboia on February 7, and, uniting with the first-mentioned high, remained stationary for a time over Montana until February 11, when a maximum of 31.42 inches was recorded. It was an offshoot of this high that moved southeastward over the Upper Mississippi Valley, the Ohio Valley, the lower lakes, and the middle Atlantic States, reaching the Atlantic coast, in the vicinity of Chesapeake Bay, on the morning of the 11th. This was a period of intense cold. No record had ever been made of such low temperatures as then prevailed.

On the 11th and 12th the high, which was stationary over Assiniboia for three days, began moving south, reached Texas on the 12th and Florida on the 13th, and thence took a northeasterly direction up the Atlantic coast; but this latter movement was not accompanied by quite such low temperatures as prevailed on the 11th.

The effects of cold were more pronounced in the Northwest, Central, and Southeastern States. The New England States and the extreme Southwest escaped.

Reports to the Weather Bureau show that 104 persons lost their lives between January 29 and February 13 from freezing and by avalanche (Colorado). The distribution was as follows: Colorado, 24; Texas, 15; Pennsylvania, 11; New York, 10; Illinois, 8; Mis-

souri, 6; Ohio, 3; Maryland, 3; Iowa, 4; Wyoming, Delaware, Virginia, North Carolina, Alabama, Arkansas, Kentucky, and New Jersey, 2 each; Washington, South Carolina, and Georgia, 1 each. The usual reports of snow-bound trains, blockades in cities, and hardship from scant food and fuel supply were more widespread than in any other cold wave of which we have records in this country. The financial loss was incalculable. One of the most remarkable phenomena was the flow of ice down the Mississippi River on the 17th of February, past New Orleans, and into the Gulf of Mexico,—an event never before witnessed during the memory of man. Ice an inch thick formed at the mouth of the Mississippi, in East and Garden Island Bays, and the temperature fell to 10° F. on the 13th. Another phenomenon was the fall of snow at Jacksonville, Florida.

Here the temperature fell forty degrees in the twenty-four hours ending at 8 A.M. on February 9, when a temperature of 10° F. was recorded, and the snow was from one to one and a half inches deep. Snow fell as far south as Titusville, latitude 28° 32′, and Tampa; at Palm Beach the temperature fell to 28°; at Miami, latitude 25° 50′, it touched 29° on the 14th.

Fish and game-birds were killed in great numbers by the extreme cold, and the loss of life among cattle on the Western plains was very great, owing to the depth of snow and the continued cold weather. Minimum temperatures of 25° below zero were observed at several places between Washington and Baltimore. The new record established for Washington was −15°, −7° at Baltimore, and −6° for Philadelphia.

The snow-fall in Philadelphia began on February 11 at 9 P.M., and continued until the 14th at 1 A.M., during which time 18.60 inches fell. The wind was from the northeast, and blew at times at the rate of sixty miles an hour. The lowest barometer was 29.92 inches. The estimated monetary loss in Philadelphia was $2,500,000, and in the country at large $20,000,000. Food rose in price, and there was a shortage in meat, coal, milk, fresh vegetables, oysters, fish, and flour. The loss to railroads in receipts and in track-clearing amounted in Philadelphia to about one million dollars. The vagaries of American climates were well illustrated in this storm. Some of the most striking phenomena were witnessed in Colorado, where several persons lost their lives by an avalanche.

On February 12 the storm had lasted without cessation for nine-

teen days in the vicinity of Leadville. At Trinidad and many other points south of Denver the mercury fell 50° F. in two hours. At Colorado Springs the blizzard was accompanied by a fall of 35° in half an hour. At Topeka, Kansas, the Government thermometer at noon on the 11th registered 18° below zero. This is unprecedented for that time of day in Kansas. Twenty-six degrees below zero was recorded in Omaha, Nebraska. In Huron, North Dakota, it was 36° below zero, and at Duluth, Minnesota, and La Crosse, Wisconsin, −32°. On account of the absence of snow, the ground in the vicinity of Chicago was frozen in many places to the depth of five and one-half feet.

# THE RELATION OF LOCAL METEOROLOGIC CONDITIONS TO THE INFLUENZA EPIDEMIC IN PHILADELPHIA, WINTER OF 1898–99.

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The rapidity and extensiveness of the spread of influenza have been its conspicuous characteristics ever since its recognition as a peculiar, if not definite and specific epidemic, disease. The very name influenza indicates more or less historic and present-day belief in the active or passive influence of the surrounding atmosphere in the production of the affection.

The problem involving a study of the meteorologic conditions relative to the etiology of epidemics of influenza is essentially a large and intricate one; for it means the investigation, analysis, and comparison of a great mass of accurately recorded data representing meteorologic observations for epidemic and non-epidemic years, throughout widely distributed localities, preceding and accompanying epidemics, and so on.

Moreover, it would be desirable and helpful to know what, if any, special weather states bear a causal relation to influenza, either in predisposing individuals to the disease, or in encouraging the cultivation of Pfeiffer's bacillus ready for active invasion, or both. In short, the problem is primarily, and, for the present, purely scientific. Will the outcome be practical, also? "We cannot, however," says Finkler, "*at the present time* (italics mine) at least, predict the appearance of an influenza epidemic from any existing atmospheric conditions." But is it not really likely that a careful study may afford the induction of principles that may have at least some hygienic value, just as storm signals, when heeded, may save lives and property?

That the idea of atmospheric conditions influencing the produc-

tion or aggravation of "grip" has been much shunted in positive and negative directions, medical history clearly shows, so that, on *a priori* grounds, there are no sound or sure premises upon which to argue a safe and stable conclusion. Schönbein, Flint, Pepper, Osler, and others, give no credence to any meteorological influence on the disease. Seminola, Goodhart, Poore, Curtin and Watson, Finkler, Assman, and a few others, on the contrary, indefinitely suspect, and, lacking evidence, more or less rationally assume the probable relation of certain meteorologic states to epidemic influenza.

To my knowledge, only within the past six months has any particular study of the subject been reported. I refer to the work of P. Dignat.[1] While my own data were being gleaned, my attention was directed to his analysis of the meteorologic conditions concomitant to influenza in Paris since 1895. He considers an abnormal increase in the barometric pressure, abnormal temperature ranges, lowering of the electrical influence, abnormal predominance of north winds, and weakening of the actinometric degree to be the meteorologic conditions that precede epidemics of influenza.

The tabulated data given below in connection with the recent epidemic are the result of merely local and limited research; and show, also, their relation to those of three previous years of milder prevalence of the affection. Hence this paper is simply a preliminary one.

The figures given were transcribed from the records made and kept in the Philadelphia station of the United States Weather Bureau; and it was through the uniform and cordial courtesy of Mr. L. M. Dey, local forecast official, and his assistants, that this work was made easy and pleasant, instead of monotonous and difficult.

Referring to the table above, it will be seen that in the recent epidemic of influenza (1898–99), the mean monthly atmospheric pressure was moderately high for the two months (October and November) preceding the marked outbreak, although quite a number of sporadic cases, especially in the latter month, were noted. During the month of rapid development (December), the pressure mean was also moderately high. The highest mean occurred in January, while the disease was most severe and generally prevalent (excepting the last week), the extremely high pressure of 30.907 having been reached on the second day of the month.

[1] Jour. de Méd. de Paris, 18me Année, Vol. x., No. 45, Nov. 9, 1898.

TABLE No. 1.

ATMOSPHERIC PRESSURE (INCHES).

| Years. | Barometer. | October. | November. | December. | January. |
|---|---|---|---|---|---|
| 1899. | Mean Monthly | . . . . | . . | . . . . | 30 134 |
| | Highest | . . . . | . . . . | . . . . | 30.907 |
| | Lowest | . . . . | . . . . | . . . . | 29.329 |
| | Range | . . . . | . . . . | . . . | 1.578 |
| 1898. | Mean Monthly | 30.096 | 30 051 | 30.034 | 29.918 |
| | Highest | 30.415 | 30.450 | 30.446 | 30.426 |
| | Lowest | 29.428 | 29.252 | 29.063 | 29.253 |
| | Range | .987 | 1.198 | 1.383 | 1.173 |
| 1897. | Mean Monthly | 30.030 | 29.994 | 29.979 | 30.021 |
| | Highest | 30.506 | 30.490 | 30.44 | 30.584 |
| | Lowest | 29.543 | 29.318 | 29.17 | 29.459 |
| | Range | .963 | 1.172 | 1.26 | 1.125 |
| 1896. | Mean Monthly | 29.940 | 30.102 | 30.074 | 30.074 |
| | Highest | 30.383 | 30.645 | 30.79 | 30.447 |
| | Lowest | 29.518 | 29.482 | 29.51 | 29.572 |
| | Range | .865 | 1.163 | 1.27 | .875 |
| 1895. | Mean Monthly | 29.942 | 30.067 | 30.028 | . . . . |
| | Highest | 30.462 | 30.548 | 30.64 | . . . . |
| | Lowest | 29.429 | 29.325 | 29.44 | . . . . |
| | Range | 1.033 | 1.223 | 1.20 | . . . . |

If the calculations mentioned by Professor Davis,[1] of Harvard, be correct,—namely, that the mean atmospheric pressure over the whole earth for the year is 29.89 inches, and for the northern hemisphere for January, 29.99 inches,—and when it is realized that increments or decrements of a few tenths or hundredths of an inch may involve changes of potential significance in the atmospheric mass, the barometric figures given above deserve careful consideration.

Comparing the mean monthly pressure of these epidemic months of 1898–99 with that of the corresponding months of 1897–98, when influenza was lightly prevalent, the diminished pressure for the latter is obvious; although the highest barometric readings for the corresponding months were about the same, except for January.

In 1895–96 and 1896–97, the mean monthly atmospheric pressures for November, December, and January were also moderate, and yet "grip" was not intense, though present to a degree. In January, 1897, this affection manifested a brief exacerbation, and we note that the barometer registered the high point of 30.584 inches (on the

[1] Elementary Meteorology, p. 92.

31st); preceded by the November and December, 1896, highest points of 30.645 and 30.79 inches respectively.

Perhaps the most marked feature is the distinctively greater absolute range between the highest and lowest atmospheric pressures for the epidemic months (December and January) of 1898–99, as compared with those of 1895–98, showing a probable relation of prevalence to extreme pressure changes, the lowest readings being (with one exception, viz., January, 1899) lower than the lowest for the corresponding months of the latter years.

TABLE No. 2.

MEAN MONTHLY TEMPERATURE.

| Months. | 1899. | 1898. | 1897. | 1896. | 1895. |
|---|---|---|---|---|---|
| October | . . . | 58.6° | 56.1° | 52.4° | 51.2° |
| November | . . . | 44.6° | 44.8° | 49.2° | 45.4° |
| December | . . . | 35.9° | 36.6° | 33.9° | 37.8° |
| January | 32.3° | 34.5° | 29.8° | 30.2° | . . . |

The mean monthly temperatures show that it was slightly colder during the epidemic months (December and January), 1898–99, than during the corresponding months of the preceding winter,—but not quite so cold as the winter of 1896–97, when influenza was again somewhat epidemic,—and, on the average, about equal in temperature with the same months of 1895–96. It is to be noted that the mean monthly temperature for October preceding the recent epidemic was rather high comparatively; as was also that of November, 1896,—in both instances about two months before the height of the influenza epidemic.

TABLE No. 3.

TEMPERATURE,—MEAN MONTHLY MAXIMA AND MINIMA.

| Months. | 1899. | | 1898. | | 1897. | | 1896. | | 1895. | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Max. | Min. | Max. | Min. | Max. | Min. | Max. | Min. | Max. | Min. |
| October | . . | . . | 66.5° | 50.7° | 66.9° | 49.6° | 60.2° | 47.0° | 61.3° | 44.0° |
| November | . . | . . | 51.3° | 37.8° | 53.1° | 38.7° | 58.3° | 42.5° | 53.8° | 39.8° |
| December | . . | . . | 42.0° | 29.8° | 44.4° | 31.8° | 41.3° | 28.2° | 46.0° | 32.3° |
| January | 39.5° | 25.1° | 41.2° | 29.5° | 37.3° | 24.2° | 36.6° | 25.5° | . . | . . |

This table gives the means of, or average differences between, the highest and lowest temperatures for the months noted, and is to be discriminated, therefore, from the absolute and greatest daily ranges of temperature for each month.

During the height of the recent "grip" epidemic (December, 1898–January, 1899), both maxima and minima of temperature were lower than for the same months of the previous winter, with a lighter epidemic; but not so low, quite, as in 1896–97, when the disease was also markedly in evidence.

An exceptionally low mean maximum for a mild influenza year is observed for January, 1896, although this is counterbalanced somewhat by the relatively much higher means, both maximum and minimum, for December, 1895.

Of the preceding autumn months of October and November nothing characteristic seems to mark the data, unless it be the rather close correspondence between the figures for the mean maxima and minima of October for the years 1897 and 1898 respectively.

Regarding temperature extremes (not tabulated here) for the month of December, the *absolute ranges* (between the highest and lowest points reached) for the years 1898–97–96–95 were 44°, 47°, 48°, and 53° F., respectively; while the *greatest daily ranges* of the same month for these years were 35°, 25°, 21°, and 28°, respectively.

For January, 1899, and the three preceding years, the *absolute ranges* were 50°, 46°, 54°, and 49° F., respectively; and the *greatest daily ranges* were 29°, 27°, 25°, and 20°, respectively. From these figures it will be evident that the absolute ranges of temperature bear no characteristic relation to decidedly epidemic years on the one hand, or to merely sporadic or mildly epidemic years on the other, unless we except January, which shows a greater absolute range in the former than in the latter. But any relative importance that absolute ranges of temperature throughout given months and years may have, in such a study as this, is at best only problematic and indefinite.

Furthermore, whilst the greatest daily ranges for the two months (December, 1898–January, 1899) of the height of the epidemic in Philadelphia show marked extremes between the maxima and minima as compared with preceding years, their direct relationship will depend most probably upon the dates of their occurrence, their sud-

denness, rapidity, and frequency of movement,—data not obtainable for this article,—with one exception: the latter shows that both the greatest daily ranges, of 35° and 29° F., occurred on December 31, 1898, and January 10, 1899, respectively, while the influenza was most violent.

However, the table above and that following (No. 4), giving the means of more extensive observations, are evidently better criterions of temperature conditions.

Finally, I found that, taking the actual number of days that the mean temperatures were above 50°, 59°, and 68° F. (U. S. Weather Bureau Monthly Records), October, 1898, was an unusually warm month compared with the four years immediately preceding. November, on the other hand, was decidedly colder in 1898, two days of mean temperature below 32°, and seven days of minimum below 32°, having been recorded. December showed great comparative coldness, too, not only in the small number of days of mean temperature above 41° and 50° (3 and 1, respectively), as contrasted with previous years, but in the fact that there was one day in which the maximum temperature was below 32°, and seventeen with a minimum below 32°; in the same way, January exhibited a distinctly, though less marked, relative increase of coldness.

TABLE No. 4.

TEMPERATURE,—MEAN DAILY RANGE AND MEAN VARIABILITY.

| Months. | 1899. | | 1898. | | 1897. | | 1896. | | 1895. | |
|---|---|---|---|---|---|---|---|---|---|---|
| | M. D. R. | M. Var. | M. D. R. | M. Var. | M. D. R. | M. Var. | M. D. R. | M. Var. | M. D. R. | M. Var. |
| October . . . . | . . . | . . | 15.8° | 3 6° | 17.2° | 5.1° | 13.2° | 3.6° | 17.3° | 4 3° |
| November . . . | . . . | . . | 13.5° | 3.3° | 14.4° | 6.0° | 15.9° | 6.8° | 14.0° | 6.4° |
| December . . . | . . . | . . | 12.2° | 4.2° | 12 5° | 4.9° | 13.1° | 4.6° | 13.7° | 5.5° |
| January . . . . | 14.4° | 6.9° | 11.6° | 4.7° | 13.1° | 6.2° | 10.9° | 4.4° | . . . | . . |

The *mean daily range* and *mean variability* afford a very fair basis for estimating the equability of temperature in various localities and at different times. The mean variability represents the

monthly mean of the day-after-day differences in maxima and minima or daily ranges of the dry bulb thermometer.

Analysis of the table shows, contrary to what one might expect, that the months of November and December, 1898, were more equable than for the other years; but that January, 1899, was exceptionally non-equable.

TABLE No. 5.

RELATIVE HUMIDITY,—MEAN MONTHLY (PER CENT.).

| Months. | 1899. | 1898. | 1897. | 1896. | 1895. |
|---|---|---|---|---|---|
| October | .... | 75.4 | 72 | 73 | 61 |
| November | .... | 74.2 | 72 | 75 | 78 |
| December | .... | 70. | 77 | 68 | 70 |
| January | 71.2 | 72. | 74 | 71 | .... |

It is usually stated that influenza prevails, by selection almost, in cold, damp weather,—rather vague terms, and totally lacking in scientific precision or rational discrimination. The history of influenza epidemics shows that the disease may spread in warm or cold, dry or damp weather, yet the rapidity, intensity, and universality of spread may be dependent in a measure upon certain sequences and associations of atmospheric temperature and moisture.

It is certainly true that the temperature of the air that we *feel* is very much influenced by the amount of vapor it contains at a given time—*absolute humidity*. But it is much more appropriate and important to determine the *relative humidity*, which expresses the percentage of saturation, or the proportion of aqueous vapor actually present compared with the amount of vapor that might exist if the air were saturated at the temperature taken.

A glance at the table above shows the average mean relative humidity for October and November, 1898, to have been distinctly higher than for 1897, and a little higher than in 1896, when influenza was mildly epidemic; and again markedly higher than in 1895, also an "off" year like 1897. The mild influenza year of 1896 also shows a higher relative humidity for these breeding (?) months as compared with the sporadic years 1895 and 1897.

On the other hand, while "grip" was at its height, during Decem-

ber and January of the recent prevalence, the mean percentages of saturation were lower than for the two preceding winters, and practically the same as for 1895–96.

Just here it should be stated that the *mean* monthly dew-points have been directly in accord with the variations of relative humidity, —*i.e.*, comparatively high in October and November and low in December and January, for the years tabulated.

As a test of the mean monthly relative humidity table, the hygrometric records (means of the daily 8 A.M. and 8 P.M. observations) for the month of December only were classified, as follows:

TABLE No. 6.

RELATIVE HUMIDITY, BY DAYS, FOR DECEMBER.

| Years. | No. of Days Percentage above | | | No. of Days at |
|---|---|---|---|---|
| | 70 | 80 | 90 | 100 per cent. |
| 1898 | 15 | 9 | 6 | 2 |
| 1897 | 23 | 12 | 6 | 3 |
| 1896 | 14 | 4 | 1 | 0 |
| 1895 | 16 | 9 | 4 | 0 |
| Means | 17 | 8.5 | 4.25 | 1 25 |

This corroborates the statement regarding the lower relative humidity for the time of greatest development during the influenza epidemic of the past winter, as well as of the beginning of the development of that of 1896–97, compared with the alternate years of slight disturbance.

TABLE No. 7.

PRECIPITATION (INCHES).

| Months. | 1899. | 1898. | 1897. | 1896. | 1895. |
|---|---|---|---|---|---|
| October | .... | 4 85 | 1.70 | 2.08 | 2.97 |
| November | .... | 7.19 | 4.44 | 2.50 | 2.32 |
| December | .... | 3.21 | 4.52 | 1.00 | 1.76 |
| January | 4.01 | 4.10 | 2 17 | 1.57 | .... |

Any possible etiologic relation that the amount of precipitation might have upon the epidemicity of influenza would seem to be connected with the soil rather than the air. And, indeed, assuming such a telluric relationship to be true, reasoning *a priori* we would infer that the influence of a dry or wet soil, as the case might be, would be a predisposing rather than an exciting one,—a good culture medium for the bacilli of Pfeiffer, as it were.

The question arises, then, whether the considerable rainfall of October and November, 1898, had any effect in preparing a rich and robust crop of influenza microbes. A relatively moderate amount of precipitation was also noted in the fall of 1896 and 1895.

It may be pertinent to this matter, also, to mention the fact that last September (1898) was dry, the total rainfall having been but 1.82 inches; while August, on the contrary, was remarkable in that the total precipitation was 9.06 inches, 5.43 inches of which fell within one hour and forty-four minutes.

TABLE No. 8.

WIND: PREVAILING DIRECTION, TOTAL MOVEMENT, AND VELOCITY (MILES).

| Months. | 1899. | | | 1898. | | | 1897. | | | 1896. | | | 1895. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Prev. Dir. | Total Momt. | Av. Hourly Vel. | Prev. Dir. | Total Momt. | Av. Hourly Vel. | Prev. Dir. | Total Momt. | Av. Hourly Vel. | Prev. Dir. | Total Momt. | Av. Hourly Vel. | Prev. Dir. | Total Momt. | Av. Hourly Vel. |
| October . . | . . . | . . | . . | N.W. | 7250 | 9.0 | N.E. | 7978 | 10.7 | N. | 7618 | 10.2 | N.W. | 7999 | 10.8 |
| November | . . . | . . | . . | N.W. | 7354 | 10.2 | N.W. | 6998 | 9.7 | S.W. | 7097 | 9.9 | N.W. | 7195 | 10.0 |
| December | . . . | . . | . . | S.W. | 7857 | 10.6 | N.W. | 7563 | 10.2 | N.W. | 7281 | 9.8 | N.E. | 8492 | 11.4 |
| January . | S.W. | 7942 | 10.6 | N.W. | 7837 | 10.5 | N.W. | 8422 | 11.3 | N.W. | 8121 | 10.9 | . . . | . . | . . |

The *prevailing direction* of the wind (from the west) was generally opposite to the direction (westward) of travel of the epidemic. Most of the time the wind blew from the northwest during the months tabulated, except those of the greatest prevalence of influenza last winter, when it was southwest. It should be noted, however, that in December, 1898, the wind blew from the northwest almost as much as from the southwest. From the northeast and east,—that is, more in the trend of spread of the disease,—the wind was observed to

blow from 14 per cent. to 24 per cent. of the time throughout the four months. See table below.

TABLE No. 9.

WIND: DIRECTIONS AND PERCENTAGES (1898–99:)

| Months. | Percentage of Times Wind observed blowing from | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | N. | N.E. | E. | S.E. | S. | S.W. | W. | N.W. | Easterly. | Westerly. |
| October | 13 | 13 | 11 | 16 | 10 | 8 | 10 | 19 | 40 | 37 |
| November | 8 | 10 | 7 | 5 | 13 | 12 | 10 | 35 | 22 | 57 |
| December | 6 | 11 | 3 | 3 | 7 | 28 | 16 | 26 | 17 | 70 |
| January | 21 | 10 | 6 | 5 | 10 | 23 | 19 | 6 | 21 | 48 |
| Totals | 48 | 44 | 27 | 29 | 40 | 71 | 55 | 86 | 100 | 212 |

Whence it is apparent at once that the westerly winds prevailed more than twice as often as the easterly, especially during the heavy "grip" month of December,—more than four times as often. Certainly influenza, unlike a United States weather map arrow, does *not* "fly *with* the wind," in its prevailing direction at least.

Nevertheless, since westerly winds are usually associated with high barometer, low temperature, and low relative humidity, it is not unreasonable to infer that in some way, by their combined effect upon either the human anatomic or the telluric soil, the disease and its germs are favored in their propagation and multiplication.

The *total wind movement*, in miles (Table No. 8), was relatively greater during the recent epidemic than during the same period of 1897–98; and also for January, 1897 ("grip" prevailing), as compared with January, 1896. But the preceding month of October shows correspondingly and considerably less total movement of wind.

The *average hourly wind velocity* was only slightly higher for the epidemic months of last winter as compared with the same months of the year previous. It is more likely that the degree of *maximum velocity* of wind at certain times may bear some relation to the spread of influenza. Thus, the weather records show that October, 1898, was unusually calm and free from high winds, as compared with former years, the maximum hourly velocity for the month having been but twenty-seven miles, which was decidedly lower than for at least

nine years previous, and this on the sixth day of the month,—some time before la grippe became epidemic.

On the other hand, not only was November, 1898, characterized by a greater total wind movement than for four years prior, but by increased high winds,—principally northwest,—above twenty-five miles per hour on the 8th, 11th, 20th, and 27th days of the month (26, 36, 28, and 42 miles per hour, respectively), while sporadic cases of influenza were beginning to multiply rapidly. The maximum hourly velocity for the month—forty-two miles on the 27th—was higher than any that had been reached for seven years.

Influenza became virtually an epidemic in the first week of December, 1898, and after the high wind just noted above came the maximum wind velocity of this month on the fourth day, from the southeast, at the rate of forty-eight miles per hour; higher, with one exception (1895), than any year since 1890. The next day the wind blew from the west at twenty-five miles an hour. Again, during the height of the epidemic the dates and rates were as follows: 27th, 27 miles; 28th, 30 miles; 31st, 26 miles, the wind coming from the northwest on the first two days, and from the north on the last day.

TABLE No. 10.

GENERAL WEATHER CONDITIONS.

| Months. | 1899. | | | | 1898. | | | | 1897. | | | | 1896. | | | | 1895. | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Clear. | P. Cloudy. | Cloudy. | Foggy. | Clear. | P. Cloudy. | Cloudy. | Foggy. | Clear. | P. Cloudy. | Cloudy. | Foggy. | Clear. | P. Cloudy. | Cloudy. | Foggy. | Clear. | P. Cloudy. | Cloudy. | Foggy. |
| October . . | . | . | . | . | 11 | 8 | 12 | 2 | 13 | 4 | 14 | 2 | 9 | 7 | 15 | 2 | 21 | 5 | 5 | 0 |
| November . | . | . | . | . | 8 | 11 | 11 | 2 | 5 | 14 | 11 | 3 | 9 | 10 | 11 | 2 | 11 | 6 | 13 | 4 |
| December . | | . | . | . | 10 | 8 | 13 | 3 | 5 | 13 | 13 | 2 | 11 | 12 | 8 | 0 | 10 | 7 | 14 | 0 |
| January . . | 10 | 8 | 13 | 2 | 5 | 13 | 13 | 2 | 10 | 7 | 14 | 1 | 10 | 10 | 11 | 0 | . | . | . | . |
| Totals for the seasons . . | . | . | . | . | 39 | 35 | 49 | 9 | 28 | 44 | 51 | 9 | 39 | 36 | 48 | 5 | 52 | 28 | 43 | 4 |

It is interesting to note that there were twice as many clear days during the epidemic months of December and January as there were for the same months of 1897–98, and fewer partly cloudy days, but

the same number of cloudy days, and one more foggy day. November, 1898, was also more sunshiny than November, 1897.

And yet, when one considers that less than one-third of the days were clear, and that the hours of sunshine are very few during the short days of the winter solstice, it seems not improbable that somehow, on the whole, "grip" likes the darkness better than the light because certain conditions for its ravages are more favorable then than at any other time of the year.[1] Otherwise, the relatively greater number of clear days during the recent epidemic is perfectly consistent with the other meteorological phenomena that seem to be associated with the spread of influenza,—namely, that cold, dry, windy (westerly) weather and sunshine go together.

Finally, attention may be called to the fact that the number of clear, partly cloudy, and cloudy days of December, 1898, and January, 1899, were exactly equal.

My personal observations during the development and height of the late epidemic, without any endeavor to obtain precise meteorological data, were generally as follows: (1) That during November and December there were several volleys of attack, as it were, in which influenza suddenly overcame numbers of people for three or four days or a week, perhaps, and then seemed to subside to a degree, to be followed by other exacerbations and remissions of invasion; (2) these attacking periods were invariably preceded by sudden thaws, "thick, ill-smelling" fogs, and relatively warm, moist, and calm weather; (3) they were apparently introduced by, and associated with, cold, clear, dry, and windy weather; (4) these changes appeared to be unusually sudden, extreme, frequent, and rapid. I was impressed particularly by the character of the early evening fogs, with their intensely carboniferous odor and suffocative heaviness. Physicists accepting the theory that each tiny mist particle has for its nucleus a particle of soot or smoke or dust, would seem to have a rational explanation for fogs; and the phrase "climate of cities" is truly fitting. It is a fair question to ask whether the sporadic cases of influenza in non-epidemic years occur only in large towns or cities and thickly populated districts; and whether their local peculiarities of weather are directly or indirectly concerned in their production.

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[1] The rare experience of a mid-winter thunder-storm was had on December 22, 1898.

As hinted at the beginning of this paper, we have to face these queries: Do weather conditions in general prepare for or sustain epidemics or pandemics? Are there special atmospheric phenomena that increase the susceptibility and weaken any possible immunity of our bodies to influenza; or that facilitate the growth and multiplication of the bacilli of Pfeiffer, or both? Or, is it more probable that meteorological occurrences influence the former more, and telluric states the latter more? *Das kommt darauf an!*

Future studies of future epidemics and, comparatively, of normal years, over widely scattered places of the "grip" areas of the globe, by careful and competent observers only will enable us to know precisely the answers to these queries.

Any summary that the data of this unfinished study may justify is practically in accord with my statements (2) and (3) given above. The conclusion of the whole matter thus far is partly interrogatory and problematic.

# Neurology.

## HEREDITARY ATAXIA.

CLINICAL LECTURE DELIVERED IN RUSH MEDICAL COLLEGE.

BY HENRY M. LYMAN, A.M., M.D.,

Professor of the Principles and Practice of Medicine in Rush Medical College, Chicago, Illinois.

GENTLEMEN,—The patient to whom I now direct your attention is a boy, sixteen years old, who has recently arrived at the age of puberty. His mother, who accompanies him, presents the appearance of ordinary health, and she asserts that her husband has been always healthy. But by this time you have learned the utter unreliability of such statements when made in the clinical amphitheatre; so we must hold in reserve our opinions regarding possible syphilis, etc. Whatever may be true of the parents, it is certain that the children are far from the normal type of health. Of the other three, one, his mother says, is in a condition probably of idiocy. The second son, now fourteen years old, is becoming ataxic. The third is in some way defective, though, as he is not before us, it is impossible to decide as to his particular form of nervous disorder.

This boy is said to have been healthy at birth. He continued to enjoy good health until seven years of age, when he passed through scarlet fever without unusual or notable symptoms. After this he remained well till his twelfth year, when he was attacked with tonsillar diphtheria. From this he made a good recovery, but soon after it was remarked that his movements were becoming ataxic. During the period of four years since that illness he has been gradually growing worse, though making no complaint of pain.

Physical examination shows at once that we are dealing with a degraded specimen of humanity. The head is small, and the jaws are disproportionately large in comparison with the cranium. The eyebrows are thick and unusually prominent on account of the thickness of the supercilliary ridges of the frontal bone. The ears are large and project laterally from the sides of the head; they lack the delicate contour that makes the ear such an ornament to the normal head. There is nystagmus involving the left eye. The nose is thick, and the lips are coarse and everted, showing that the patient is a mouth-breather. The expression of the countenance is listless and dull; the voice is rough and harsh. When we cause him to bite strongly on a stick, we find that the masticatory muscles on the left side of the mouth are considerably weaker than those upon the right side. In fact, the whole left side of the patient seems weaker than the other.

Descending to the thorax and upper extremities, we find that the left serratus muscle fails to contract with normal vigor, and the scapula, therefore, hangs off from the back, to which it should be closely applied. On the right side apposition of the scapula is normal, but the supraspinatus and infraspinatus muscles are so far shrunken as to leave a decided furrow above and below the scapular spine. This also hinders the rotatory movements of the upper end of the humerus in its glenoid socket. The mid-dorsal portion of the spine is laterally bent, with the convexity of the curve towards the right, being an exaggeration of the slight normal curvature that obtains in normal right-handed subjects. The lower spinal muscles and those of the trunk do not exhibit any appearance of atrophy. The same is true of the muscles in the lower extremities, until we reach the feet, when we find that in each foot the balance between the interosseous muscles moving the second toe and its metatarsal bone is destroyed, so that the proximal and distal phalanges are in a condition of hyperextension, while the middle phalanx occupies the position of flexion.

Sensation, so far as the ordinary tests can be applied to a subject so deficient in intelligence, appears to be everywhere present, but the muscular sense is evidently lacking, for the patient cannot stand with his eyes closed, and he uniformly assumes a straddling position for his legs when trying to balance himself upon his feet and to walk. It is also impossible for him with his eyes closed to place either fore-

finger quickly upon the end of his nose or to make the forefingers meet when rapidly approached in the dark. Ataxia is thus shown to be a widely generalized phenomenon in this case.

Asking the patient to extend his arm, he does so and holds it extended without tremor. Placing a glass of water in his hand, he is able to carry it to his mouth when his eyes are open, and to drink from it without spilling or specially agitating its contents,—there is no "intention tremor."

On removing his trousers it is apparent that the lower extremities suffer from imperfect circulation of the blood; the legs and feet are cyanosed. This does not appear to be due to cardiac or pulmonary disease, but is a vaso-paralytic phenomenon occasioned by defective action of the vasomotor nerves. The knee-jerk is absent on both sides, and no amount of "reinforcement" by the method of Jendrassik, or by any other method of increasing muscular tone, will avail to elicit any response whatever to a tap on the tendon below the patella. Neither is there any "ankle clonus" when we attempt to awaken reflex contractions in the flexor and extensor muscles of the foot. The "light reflex" in the pupillary muscles is slight and imperfect, but not yet wholly abolished.

Such, then, are the salient features of this rare disease. During the last ten years we have seen only two similar cases at this clinic. Looking up the literature of the subject during the last two years, I could find references to less than three hundred cases; so you see that we have before us a pathological curiosity. First described by the German pathologist Friedreich, it was for a number of years known as Friedreich's disease, but now it is called hereditary ataxia. It owes this name to the fact that it presents certain marks of tendency to occurrence in family groups where heredity may be supposed to influence its development. In one case that came under observation in this clinic a number of years ago, the patient, a young man of nineteen, was the child of syphilitic parents who had acquired syphilis before his birth. Neither one of them was tabetic, but their son was a striking example of hereditary ataxia. Whatever influence syphilis exerted in the evolution of his disease was derived from his parents. I was unable to learn from his mother that he, when a child or later, had ever experienced syphilis itself.

The tendency to occurrence under the influence of family causes is illustrated in the present instance. One of his brothers is already

ataxic; another is idiotic; still a third, whom we have not seen, is nervously or mentally defective.

I have called your attention to a causal relationship between this disease and antecedent syphilis. It cannot be considered a direct consequence of syphilitic infection; but, as in true dorsal tabes, when the tissues have been long soaked in poisons of microbic origin, the spinal cord is rendered more liable to yield to the common causes of sclerosis than would have been otherwise the fact. Accordingly, we find that it occurs sometimes after the ordinary infective diseases, such as measles, scarlet fever, diphtheria, influenza, smallpox, etc. It is one of the occasional sequences of a hard-drinking ancestry. In this respect alcohol, the product of microbic life, seems to predispose the tissues to disease just as the analogous products of other infective microbes do,—just as the toxins of parental syphilis appeared to affect the tissues of the boy of whom I spoke just now. Like many other diseases involving the growth and nutrition of the body, it is first encountered during the period of first dentition and at the age of puberty. It is rather more frequent among males than among females; yet the disproportion is not so large as in tabes dorsalis.

Conspicuous among the symptoms of the disease is the unsteadiness of gait which gives it the name of ataxia,—hereditary ataxia. This phenomenon is usually manifested first in the lower limbs,—sometimes in all the extremities at once. There is rather less titubation than in ordinary tabes; the patient straddles and staggers as if tipsy, or like the victims of cerebellar disease. The muscles of the neck are involved, so that the head waggles on the shoulders. The eyes cannot be kept quiet, but are often affected with movements of nystagmus; and the muscles of articulation are incapable of co-ordination, rendering speech irregular, tardy, stammering, and indistinct. The eye symptoms of tabes dorsalis—pupillary rigidity, loss of light reflex, and atrophy of the optic nerve—are almost always absent, though in the present instance you see that the pupils do not respond as readily as they should to variations in the light, and the muscles of the left side of the face are weaker than those on the right. The muscles that move the eyeballs are not paralyzed, though the existence of nystagmus indicates some disorder of the central motor apparatus for the eye.

Hereditary ataxia differs also from ordinary tabes in the fact that

sensory disorders are not of common occurrence. There are none of the characteristic lancinating, burning, boring pains that agonize the tabetic patient; and the visceral crises of headache, laryngo-tracheal spasm, cardialgia, gastralgia, enteralgia, and genito-urinary neuralgia that are so often experienced by tabetics are quite unknown in this disease.

Trophic disturbances are not common, though present in certain muscles in the case before you; but it is usual to meet with deformities of the spine and various forms of club-foot, caused either by weakness or atrophy of some of the muscles that support the spine and move the extremities. The deformity, as in this case, usually consists in a lateral curvature of the thoracic portion of the spinal column; occasionally it assumes the character of antero-posterior curvature, either backward (kyphosis) or forward (lordosis). True muscular atrophy is usually witnessed in the later stages of the disease, so we may in this instance infer a rapidly progressive course of degeneration, involving the central gray matter in the anterior cornua of the spinal cord.

It is not unusual to discover in these cases evidences of disturbance of secretion; there may be excessive perspiration, salivation, or polyuria. Vasomotor defects are frequent, and in this patient are indicated by the cyanotic appearance of the lower limbs. Sometimes epileptiform convulsions are witnessed, and usually the mental faculties are obtunded, so that the child is considered backward or idiotic. The patient before you does not exhibit any peculiar movements aside from those of simple incoördination, but the other case to which I have alluded was decidedly choreic; and in one instance I have seen the peculiar movements of athetosis.

The anatomical changes that underlie these symptoms are chiefly encountered in the posterior and lateral columns of the white matter of the spinal cord. On opening the spinal canal the pia mater over the posterior or dorsal surface of the cord is often found thickened as if by chronic subacute inflammation. The entire thickness of the cord is also reduced as if by a shrivelling of its tissues. The actual change consists in a degeneration of the fibres and cells that make up the true nervous substance in the posterior white columns, the ascending cerebellar columns, the lateral pyramidal tracts, and the columns of Clarke. The root zones along the inner margin of the posterior cornua of gray matter also participate in the degenerative process.

The neuroglia in all the affected parts is greatly multiplied, and by its proliferation the true nerve substance is compressed and smothered out of existence. In certain cases—evidently in this one—the central gray matter in the ganglionic foci of the cornua is in like manner invaded and subjected to degeneration, so that the muscles which happen to be connected with such ganglionic groups cease to receive trophic guidance from the cord, and fall into a condition of progressive muscular atrophy. It is in this way that we may explain the partial atrophy of the muscles about the shoulder-joint in the case before you. Sometimes the degenerative process also invades the higher nerve-centres,—as, for example, the cerebellum,—and, doubtless, in the present instance the motor root of the fifth nerve that gives branches to the muscles of mastication on the left side of the face has suffered in the same way. It is in their posterior spinal nerve-roots that degeneration usually attacks the peripheral nerves, but occasionally many of the longitudinal nerve-fibres have been found destitute of ensheathing substance as if undergoing a process of degeneration and atrophy.

Various explanations of the origin of the disease have been set forth, but they all leave much to be desired. The connection with antecedent syphilis that is so remarkable in tabes dorsalis is not clearly apparent in this disease. The number of male patients does not considerably exceed that of the female subjects of the disease, whereas in diseases of a syphilitic origin the number of males is usually four times as great as that of females. But it seems evident that the origin of the disease is closely connected with a previous intoxication of the nervous tissues. Chronic alcoholism, syphilis, and insanity have been noted as parental antecedents, and various infective diseases experienced by the patients themselves have usually antedated the appearance of ataxic symptoms. Now, we know that all infective microbes produce toxic substances that, like their analogue, alcohol, exert a very injurious effect upon nervous tissue. Witness, for example, the neuralgia, the paresis, and the cerebral disturbances that are so frequent after an epidemic of influenza. Were it not for the antitoxic power of the tissues themselves, such events would be far more common than they are; but in the vast majority of cases the resistance of the nerve-cells increases when exposed to toxic invasion, and complete recovery from the infection takes place. When the nerve-cells do not thus rise to the occasion they degenerate, and,

if not actually destroyed, they live in a more or less permanently shrivelled and dying condition.

That some such explanation of the degeneration that is witnessed in tabes dorsalis is near the truth is rendered probable by the fact that in tabes the degenerative process first appears in the immediate vicinity of the vessels that nourish the tissue. But, if the toxic substances are conveyed by the blood, why do not all the nervous tissues simultaneously suffer alike? Evidently, the tissues themselves differ in their power of resistance to the toxic agent that reaches all alike. Significant in this respect is the fact that the recipient paths that convey impressions from without are the first to suffer. It is in the later stages of tabes dorsalis and hereditary ataxia that the motor regions of the cord begin to yield. The sensory apparatus of the body is more unstable and impressible than the motor; consequently, it is in the sensory functions that toxic influences are first experienced. Witness the progress of acute alcoholic intoxication: sensation is first disturbed, then the vasomotor, secretory, and trophic functions; finally the power of locomotion is inhibited.

After all, however, it must be admitted that we cannot yet furnish a satisfactory explanation of the peculiar modes of incidence of the degenerative process in such diseases as multiple sclerosis, tabes dorsalis, hereditary ataxia, alcoholic paralysis, diphtheritic paralysis, acute anterior poliomyelitis, progressive muscular atrophy, and the different paralytic phenomena that follow other infective diseases. Evidently, there is a reason for the selection of the posterior columns in tabes and of the peripheral nerves in chronic alcoholism, but we do not yet see clearly into the nature of that selective process.

Recognition of hereditary ataxia is not usually difficult. From tabes dorsalis it is distinguished by the age and history of the patient, by the absence of lightning pains, by the presence of nystagmus, by the peculiar staggering gait, and by the deformities that are frequently developed. From ordinary multiple sclerosis it is distinguished by the fact that it is a disease of early life instead of middle life, and that the knee-reflexes are absent instead of being exaggerated. The so-called "intention tremor" is lacking, and the voice in articulation is harsh rather than "scanning."

The life of these patients is usually shortened; rarely do they enter the fourth decade of existence. Death is preceded by progressive loss of muscular vigor, often reducing the victim to a hopelessly

bedridden condition, in which some intercurrent disease proves fatal.

The treatment of this disease must not be overlooked, despite the fact that it is not curative. The deformities that tend to arise from spinal curvatures may be met by the use of appropriate orthopædic apparatus. Fortunately, pain is not a conspicuous symptom, so that it is seldom necessary to prescribe the various anodynes that are often useful in the painful crises of tabes dorsalis. When there is a history of parental syphilis, mercury and iodide of potassium should be thoroughly administered for a time, as in cases of hereditary syphilis. But this treatment should not be indefinitely extended. Nerve-stretching has been recommended, but is now almost entirely abandoned. The same thing is true of suspension, after the method of Charcot in the treatment of tabes dorsalis. If employed, it should be practised only in uncomplicated cases, where there are no serious visceral or skeletal diseases.

When muscular atrophy is progressive, electricity should be employed, preferably the faradic current for ten minutes daily. If galvanism is used, the current should not be strong enough to cause pain. It is probable that the effect of galvanism and of static electricity is largely due to suggestion, and it proves inefficient when the patient has become accustomed to its use, or is without sufficient intelligence to be aroused by its action to a state of expectant attention.

In the later stages of the disease, good nursing and a comfortable bed will be the most efficient prescription that can be made.

# PARANOIA.

CLINICAL LECTURE DELIVERED AT THE HALIFAX MEDICAL COLLEGE.

BY GEORGE L. SINCLAIR, M.D.,

Late Medical Superintendent Hospital for the Insane, Halifax, Nova Scotia.

Gentlemen,—Before showing you the two cases of paranoia I will give you very briefly their histories.

A. B., aged thirty-six years, bachelor. Family history is not good. His father has for a long time been considered as peculiar, his sister died insane, and a brother is insane at this time. The patient evinced no special peculiarities until he was twelve years of age, when he began to learn the trade of a printer, and, as is so often the case with printers, he became a great reader. He was unsociable and conceited, and finally, on account of his "big head," he was discharged from the office. He returned home and learned the trade of a potter, which his father followed. He was a good workman, but irregular in his hours of labor, and rather despised his means of gaining a living. He was from this time on a somewhat morose and most unsociable man.

My first knowledge of him came from letters in which he expressed a wish to consult me as to a means of getting rid of certain persecutors he had, who, by means of electrical apparatus, were using him as a telephone post, directing his thoughts, reading his mind, forcing gases into him, tampering with his food, and acting generally so as to make his life anything but comfortable. Subsequently he called to see me and repeated at greater length his complaints. He added the intelligence that if he could discover the man "running the dynamo" he would kill him, and so get relief. He denied using liquor, but confessed that in early life he had practised masturbation. I talked to him, told him his ideas were all wrong, gave him some bromide of potassium, and advised him to try and give up the notions.

I heard no more from him for some months, when I again re-

ceived a deluge of letters, from the tenor of which I concluded that he was getting worse. His conduct at home was so strange, his assertions regarding his persecutors and their acts were so outrageous, and his threats as to what he intended doing so alarming, that every one was frightened, and, being convinced that he was insane, two doctors were called in, who certified to this fact and he was sent here. He resisted coming, and has never ceased to protest against the outrage committed on a "sane, sensible man." Of late he has been threatening me with the terrors of the law for detaining him, and has written many letters to judges, lawyers, magistrates, clergymen and editors detailing his troubles and persecutions and protesting his perfect sanity. He has not as yet fully decided as to who is to blame for the bodily and mental sensations of which he complains, but he has no doubt as to the reality of them. He rather suspects that I know and will not tell. He is an intelligent man, and knows a little about many things, but only in a superficial way, with the result that his deductions are generally wrong. He is surly in manner, solitary in his habits, and most indifferent in regard to cleanliness and neatness. He has been a patient here nearly two years, and is worse than when he came in. He still thinks that his thoughts are stolen, that his food is tampered with, that he is inflated with bad gases, and that his semen is drawn from him at night. He frequently uses the most horrible profanity and threats towards his imaginary persecutors.

I will now let you see him, and possibly he may tell you of some of his troubles. (The man came in, but positively refused to say anything.) I am not surprised at his silence. He has a fair amount of self-control and is suspicious of the intentions of others, especially strangers.

The other case is that of C. D., aged thirty years, a bachelor, temperate, masturbator. Family history good. Parents healthy, both living. Two sisters and a brother quite well. No nervous strain, as far as I can find out.

As a boy he was always of a shy, retiring disposition, distinctly unsociable. As a youth he learned the trade of a tailor, and was regarded by his fellow-workmen as peculiar. He was constantly suspicious of others in regard to their actions toward him. He started business as a tailor and in a short time failed. Then he went to keep the books of his brother, who was proprietor of a large country store. His manner to all was now most disagreeable, and he developed delu-

sions in regard to certain customers which resulted in his losing his situation. His delusions became distinctly those of persecution. He thought some one was running electricity through him, to his vital and mental injury. He said his stomach was being pumped full of gas and his genitals were "toyed with" and his semen drawn off. He was suspicious that his food was poisoned, and thought that he had "a priest in his stomach and a chapel in his head." This idea he still holds. He has visual and aural hallucinations. The latter are the most distinct and occur sometimes in one ear, sometimes in both. The voices suggest lewd ideas and use "swear words." At meal-times especially he is much tormented, as he hears remarks about the quality of his food which are most disgusting. When he sits at table he digs his fingers into his ears to clear them of the sounds which make eating well-nigh impossible. At night he can be heard pleading with the priest in his head to let him alone; the next moment he will use the most horribly profane language. He has nocturnal emissions, and lays the blame of this upon certain persons whom he accuses of masturbating him and of practising sodomy upon him. He has reached the stage in his disease when he blames individuals rather than societies for his persecutions, and he not unfrequently tells me that some one of his acquaintances is responsible for a particular annoyance. He is not in good general health, but is anæmic and constipated.

You can now see him and question him upon his symptoms. (The patient spoke freely of his delusions, and also stated that he would not hesitate to kill certain persons, if he met them, as a means of freeing his life from its present miseries.)

Having let you see these two cases of paranoia, I will now give you a brief account of the disease.

*Paranoia.*—This affection resembles the disease formerly called monomania or delusional insanity, and may be defined as "a progressive, systematized mental disorder, chronic in its course, without derangement of the general health, and characterized by hallucinations, especially of hearing, and by delusions which are almost invariably those of persecution."

In a typical case there are usually certain positive features. There is a history of hereditary traits and some eccentricities evinced during childhood, more marked peculiarities during youth, and often associated with hypochondriasis. About the age of thirty years system-

atized delusions of persecution appear, which later are combined with, or give place to, those of an exalted character, religious, patriotic, philosophic, or erotic. But, while these delusions may dominate mental action, they need not impair every faculty.

The early symptoms, those of hypochondriasis, startle and alarm the patient and lead him to concentrate his attention upon himself. He has various new and uncomfortable feelings, such as headache, palpitations, buzzing in his ears, and vague, uneasy sensations about the alimentary tract or in the genital region. With these there is an ill-defined dread that the intelligence is being overthrown and that he is losing the power of thinking or of controlling his thoughts.

Naturally he searches for a cause for his trouble, and, unlike the ordinary hypochondriac, he does not look so much inside as outside himself for it. Not finding one, he becomes morose and irritable. As time passes the introspection continues, and the patient is prone to think that all he sees and hears has reference to him. From his new point of view everything is changed. He imagines that people passing look at him peculiarly or smile or jeer; what he hears has a double meaning, and the horror of his position becomes so impressed upon him that he may even contemplate suicide; this is rare, however, and generally he accepts his fate, but continues to seek a cause for his sad and changed state. He reviews his past life with painful minuteness, and convinces himself that he has all along been an object of animosity to some forces or influences. Hallucinations of hearing make their appearance and add to the misery and discomfort. Ultimately every special sense becomes involved, to the increase of his wretchedness.

Delusions next appear. At first they are confused, but always of a persecutory type, and it was from this fact that Leseque, who has studied the disease in a masterly manner, gave it the name of persecutory insanity. In time the patient decides that he is subject to ill-will, but does not at first refer this to any one in particular. In speaking of his trouble he will say: "They electrify, poison, masturbate, sodomize me; they throw bad smells on me." As time passes he becomes more definite in his charges and blames the freemasons, the Jesuits, and the police collectively. If you ask him how such things can be, he will accuse his enemies of using strategy or invisible means to accomplish their ends. He will tell you that holes are bored in the door of his room through which foul odors are thrown upon him,

or acoustic tubes are led near him through which insults are poured into his ears, or electrical apparatus is used, or spiritualism or hypnotism or other occult agents. The explanation is satisfactory to him, no matter how unconvincing it may be to you.

As time passes the aural hallucinations increase. The voices which jeer at and insult him are heard night and day, anywhere and everywhere, sometimes in only one ear and sometimes in both. Occasionally there are two which oppose and contradict each other, or one is soothing and the other insulting. They interrupt him in an ordinary conversation, criticise what he says, and as soon as an idea is formed in his mind it is at once uttered, in spite of his wish to control it, and thus others against his will are made aware of his inmost thoughts. This is the "echo voice," a most painful and disagreeable symptom. Almost invariably he blames electrical force for this. Sometimes the patient says he thinks he recognizes the voice as that of an acquaintance or friend.

If visual hallucinations exist, they are not as sharply defined as the aural, and appear as fogs, or shadows, or influences which are not disagreeable, though unkind in their intentions.

Smell and taste are almost always affected, and may cause the patient, on account of the fancied presence of some noxious material in his food, put there by enemies, to refuse to eat at all or only of certain articles and at particular times. Painful bodily sensations are frequently present, such as spasms, twists, cramps, and burning pains. It is not uncommon to have complaints made that the stomach has been torn out or the bowels distended with gases, or that the semen is drawn from him, that he is sodomized or masturbated at night, and that lewd women get into his bed against his will and have connection with him during sleep.

At this stage the patient is usually in despair and may appeal to the civil powers for protection. Generally he writes letters to the governor, the mayor, a magistrate, or the chief of police, singly or collectively, calling attention to his case and demanding their aid in arresting his persecutors and protecting his life. Failing to obtain relief, he may change his place of residence, even going to another part of the country, only to find that in time his enemies follow him and still torment him. Finally, in sheer desperation, he decides to defend himself, and from being the persecuted one he becomes himself the persecutor. It is at this point in his career that the interest

of the general public begins. There is no more dangerous person to be at large than a paranoiac. The larger number of crimes committed by the insane, both inside and out of an asylum, are the work of lunatics of this type. One can be in no greater peril than to be taken by one of this class of lunatics for the head of the conspiracy which he blames for all his sufferings and for the person against whom he must avenge himself. The danger is all the greater since the possible victim is ignorant of it, and the aggressor, in full possession of his mental resources, brings to the service of his enmity marvellous astuteness, cunning, and cruelty. At a time when he least expects it, when everything is peaceable and tranquil, one may find himself attacked by a person whom he does not know, has probably never seen, and to whom he can have done no wrong whatever.

Sometimes a paranoiac may not have his imaginary persecutor definitely fixed in his mind at all; but on the prompting of an aural hallucination, or as a morbid impulse, may attack the first person he meets. Equally with the epileptic the paranoiac is a most dangerous man, and his acts are of the same apparently spontaneous origin and violent character.

*Course and Termination.*—The course of the disease is essentially chronic, though there may be remissions or the character of the delusions may change. For instance, such a lunatic may argue with himself thus: "If I am singled out for persecution it must be from some cause. Perhaps I am more than an ordinary man, and this conduct on the part of others is instigated by jealousy." With this delusion as a starter, he easily arrives at the conclusion that he is a great political genius of whom others are envious, or that he is of royal birth or even of divine descent, and he may endeavor to lead a life in accordance with this new idea and in keeping with his illustrious personality as he conceives it.

When the patient is a woman, at this stage the change may be influenced by erotic aspirations. She may regard herself as the intended bride of the Most High, and may claim to be with child by the Almighty.

Married men have imagined that as a tribute to their greatness their wives are the recipients of improper attentions from other men, and, suspecting their fidelity, misinterpret the most innocent acts and even assault and punish them severely for imaginary infringements of their marriage vows.

If under custodial care, all these cases pass ultimately into a condition resembling dementia. It may be long in coming, but it does arrive sooner or later. During this condition some of the delusions of persecution remain and continue to influence the patient's life and actions; but the edge has been taken off of them and they are not so apt to appear in the ordinary life of the person and are not revealed in his common conversation. Very rarely distinct amelioration occurs, but a relapse almost invariably follows, with return of nearly all the original delusions.

The paranoiac is not an attractive patient. He is apt to be irritable, surly and moody, short of speech, laconic in his answers, and suspicious of the action of others towards him. Sometimes he will not reply to questions at all, or will say, "Why do you ask me this? you know better than I do," an insinuation in some instances that you can read his thoughts. He may be a prolific writer of letters in which he states his complaints, demands his liberty, and threatens those in authority with punishment if he is not released from custody. He denies any allegations made against him as a dangerous person to be at large, and will even repudiate the authorship of letters in which he has stated his grievance and threatened to have revenge.

In the ward he may be noticed sitting apart from the other patients, talking to unseen persons, and at meal-times he will stop his ears to keep his enemies from worrying him or suggesting things about his food. He is terribly in earnest in his endeavor to free himself from the influences which interfere with his eating, and he will pull at his ears and go through the motions of throwing from him his persecutors.

*Treatment.*—There is nothing to be done save to put such people where their chance of injuring others is reduced to a minimum. Custodial care is, I think, necessary, and then attention should be paid to the general health. These patients are, as a rule, not inclined to take medicine, which they either suspect is more poison or refuse because they are not in need of drugs. While under care they must be watched to see that they do not obtain a weapon with which they can do themselves or others harm.

The men, in my experience, are masturbators, and are always worse after a series of emissions. In such cases a pretty free blistering of the foreskin with liquor epispastica does good.

# IDIOPATHIC MUSCULAR ATROPHY; PERONEAL TYPE OF ATROPHY; SPECIFIC BASAL MENINGITIS.

CLINICAL LECTURE DELIVERED AT THE MASSACHUSETTS GENERAL HOSPITAL.

BY G. L. WALTON, M.D.,

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Gentlemen,—The first two of the cases I have to show you to-day have sufficient in common to assist us materially in our study of the differential diagnosis between the various types of atrophy; the other case has nothing in common with them, but is in itself one of considerable practical interest.

I shall try to bring out the salient points in these cases with a view to diagnosis, without going into detail or taking up the pathology of the conditions illustrated.

## IDIOPATHIC MUSCULAR ATROPHY.

This young man offers a striking illustration of the selective tendency of the disease known as idiopathic, in distinction from spinal, muscular atrophy. The separation of the varieties may not be of vital moment as regards treatment, medicinal effort availing nothing against either, but the prognosis offers a practical reason for care in the diagnosis. The comparatively rapid advance of the spinal variety, with a tendency to early involvement of the bulbar nuclei, compels a far more serious prognosis than the slow progress and comparative immunity (as regards life) of the idiopathic type.

The two most important diagnostic indications of the idiopathic form, according to Gowers, are the affection of more than one member of the same family and the onset of the disease before adult life is reached, the former being the more conclusive of the two. In this young man the symptoms began before the age of twenty, and he tells us that a brother of twenty-one has of late noticed a projection

of one shoulder-blade and difficulty in raising the arm, but no wasting. Other than the possibility that this is a similar affection there is no question of family tendency.

Perhaps the next most important diagnostic feature of the idiopathic form is the wasting of the zygomatici, evident in this patient at a glance. The form of spinal atrophy known as amyotrophic lateral sclerosis we may almost exclude, as he enters the room, by the entire absence of spasticity in the gait.

Fibrillary twitching and increased mechanical irritability of the muscles would tend to favor the spinal lesion. As regards the former the patient describes an occasional twitching in certain fibres, but this has not yet appeared under our observation. Mechanical irritability we shall not find increased, except perhaps moderately in the deltoids. Up to this point, then, we are not able to establish the diagnosis, further than to say that positive indications of spinal involvement are absent, on the one hand, while, on the other, the onset was early and the zygomatici are affected.

The next step is to study the groups of muscles involved, and to note if the distribution conforms to either of the well-marked types.

The best-marked variety of idiopathic muscular atrophy is the so-called "facio-scapulo-humeral" of Landouzy and Déjerine. In this form the zygomatici and the upper arm and shoulder muscles (with the exception of the deltoid and infraspinatus) receive the brunt of the lesion, the thigh muscles also becoming involved, in preference to those of the calf. The forearm escapes, excepting the supinator longus. The intercostals are apt to be involved, as well as the diaphragm.

Now we shall find that the distribution in this case coincides so closely to the facio-scapulo-humeral type that we shall be justified in classing the disorder as idiopathic rather than spinal, rendering the prognosis far more favorable than would otherwise have been the case. This subject is of sufficient practical importance to warrant our making a detailed study of the affected muscles.

The first peculiarity which attracts our attention is the gait. There is a decided toe-drop on both sides, most marked on the right. In carrying the right foot forward he raises it high, to enable the toe to clear the ground. This tendency towards flail-like relaxation of the parts (*Schlotterbeweglichkeit* of the Germans) forms a striking contrast to the spastic gait resulting from lateral sclerosis, and indi-

FIG. 1 – Facio-scapulo-humeral type of idiopathic muscular atrophy, showing wasting of upper arm muscles, intercostals, and zygomatici.

FIG. 2 —Shows projection of scapula due to paralysis of serratus magnus.

cates that the lesion has invaded either the anterior nerve-cells in the cord or the peripheral nerve-fibres on their way from these cells to the flexor muscles, or that the muscles themselves have been deprived of their function. I have already impressed upon you the general rule that in long-standing cases wasting and relaxed paralysis point to anterior horn or peripheral disease; while rigidity and so-called spastic paralysis, without marked wasting, point, as a rule, to the pyramidal tract, either in the brain or cord. This case offers no exception to the rule, not even suggesting the combination of both lesions frequently seen in the spinal type of progressive muscular atrophy (amyotrophic lateral sclerosis).

We next note the difficulty he finds in raising the arms at the shoulders, and satisfy ourselves by passive movement that this disability is not due to mechanical restriction.

The next striking feature is the wasting of facial muscles: marked hollows are seen in the region occupied by the zygomatici, and the lip seems somewhat drawn forward and upward in the central line, through the inability of these muscles to hold back the corners of the mouth. On inserting the finger in the cheek and asking him to close upon it, we find some weakness in the orbicularis oris.

The patient can wrinkle the forehead, lift the nose, and close the eyes perfectly. There is no trouble in swallowing or in speech,—in other words, no implication of the muscles supplied by the cranial nerves except the zygomatici and orbicularis oris.

The tongue is protruded in the median line and shows no atrophy. This is characteristic of the idiopathic form, the tongue being almost invariably implicated in the spinal form when the face is affected.

The eye movements are unaffected, the pupils normal. The head is moved freely in all directions, the only peculiarity of the muscles of the neck being the prominence of the sterno-mastoids on inspiration, a phenomenon due to the paralysis of the intercostals and the diaphragm, to be noted directly.

On removing the clothing a striking picture is presented. The muscles on both upper arms are greatly wasted, except the deltoid and the infraspinatus. The arm is raised to the horizontal (deltoid), but further elevation is difficult. It will be remembered that the serratus magnus plays an important part in raising the arm above

the horizontal line, and a view from the back shows that this muscle is paralyzed, the scapula being tilted and the lower angle extending backward (angel-wing paralysis). An approach to this malposition of the scapula is sometimes produced mechanically and sometimes mistaken for paralysis of the serratus; a faulty habit of drooping the shoulders, for example, produces a slight degree of this deformity; a still more marked form appears in lateral curvature of the spine. In case of suspected mechanical fault we have only to ask the patient to throw the shoulders back and approximate the shoulder-blades; this our patient cannot do, showing that we have to do with a true paralysis.

The infraspinatus is enormous, probably on account of overuse, and presents a marked contrast with the wasted muscles of the neighborhood. The biceps, on the other hand, is reduced almost to nothing, the triceps is markedly weakened, and the supinator longus is almost wanting in the left arm and nearly so in the right. This is best tested by asking him to flex the forearm with the radial side uppermost, the observer meantime holding the wrist down forcibly; in health this movement causes the supinator longus to stand out prominently, while in his case the opposition of my little finger suffices to prevent flexion. The latissimus dorsi and pectoralis major are wasted. These muscles are apt to escape notice, but are important, and best tested by holding the arm away from the side and asking the patient to draw it to the side. The muscles of the forearm and hand, with the exception of the supinator longus, are unaffected.

Inspection of the trunk shows decided flattening of the upper part of the chest from before backward. Marked wasting of the intercostals is noted, and the abdomen remains immovable during respiration (paralysis of the diaphragm); it is, therefore, apparent that the accessory muscles of respiration are performing practically the entire labor.

Proceeding to examine the lower extremities, we find a flattening of the left buttock with flaccidity of the glutæus on that side. The hamstrings are rather weak, but not markedly wasted; the quadriceps femoris is decidedly wasted and feeble in each leg, and the knee-jerk practically wanting through absence of this muscle. Abduction and adduction of the thigh are free. In marked contrast to the wasting of the thighs, the calves are large and well rounded, the

only muscles wasted below the knee being the tibialis anticus. This muscle is only so far affected on the left leg that the elevation of the toe is slightly impaired. The right toe cannot be raised at all, but he tells us that the extreme weakness of the right foot appeared only a few days ago after kneeling at his work steadily for an hour, numbness and prickling developing at the same time; it is fair to assume, therefore, that this apparent accession of paralysis was due to peripheral pressure, and to expect that it will speedily disappear, the right foot recovering the degree of power it possessed before this mishap. The possibility, however, must be recognized that this slight trauma sufficed to cause an extension of the original process.

All the superficial reflexes are wanting. Further physical examination elicits nothing of note.

Detailed study of muscular grouping in this case has been no waste of time, for the result has given us an opportunity to study a classic case of the facio-scapulo-humeral type of idiopathic muscular atrophy. The patient is also the gainer by this study, as we are enabled to assure him that his case belongs to the less active form of the disease. The bulbar nuclei will not become affected, as they almost inevitably would be in the spinal type. The bladder and rectum will not be affected; in short, the disease in itself will not directly menace life, and is likely at any time to come to a prolonged standstill.

The most troublesome feature would naturally be the involvement of respiratory muscles, but the case shows well the conservative power of nature over the vital processes, for he tells us that he has experienced no material difficulty in breathing.

The best illustration I have seen of the slow progress and hereditary tendency in this affection was in the case of an elderly patient (seventy years of age, if my memory serves me right) in whom the wasting of the idiopathic form first appeared before the age of thirty, progressed almost imperceptibly, and in no way threatened life. In that case the hereditary tendency was well illustrated, for he was able to trace the disease back through his ancestry to the landing of the Pilgrims.

## PERONEAL TYPE OF ATROPHY.

This case, which I take the opportunity of showing you in connection with the last, illustrates the difficulty of absolute classification of these atrophies. Arguments might perhaps be adduced for

presenting this patient as a case of chronic poliomyelitis, of progressive muscular atrophy, or of neuritis. I am inclined to class it under progressive muscular atrophy of the peroneal type, a disease of which the pathology is not yet established, though the clinical picture is fairly constant.

The history is, in brief, that this young woman (about twenty-four years of age) suffered from gradual loss of power and wasting in the muscles of the right leg about four years ago; the wasting progressed for six months and then remained stationary. About a year ago the left leg became similarly affected during six months. For the past six months no change has occurred. There has been no pain, no tenderness, and no definite numbness. The arms and hands are normal, and the patient in other respects is perfectly well.

The physical examination shows marked wasting of the peroneal muscles and of the tibialis anticus in both legs. The temperature of the legs is lessened, the feet are clubbed, and the toe-drop is so considerable as to impair the gait materially.

I will here remind you that anterior poliomyelitis in the adult has just this onset,—namely, subacute (from ten days to a month) or chronic (from one to six months); and if the lesion in this patient had been limited to the right leg, we should have had little hesitation in classing the case under that head. A practically symmetrical attack on the other side would offer, however, a sufficiently unusual coincidence to lead us to look further for a diagnosis.

Cases have doubtless been classed as anterior poliomyelitis that should have fallen under the head of progressive muscular atrophy. The condition of the affected parts is much the same in the two diseases, the clinical distinction depending on the course. If the disease progresses instead of coming to a stand-still, progressive muscular atrophy must be seriously considered.

In the so-called Charcot-Marie type of muscular atrophy the peroneal group is first invaded, other muscles of the lower leg follow, the extensors before the calf muscles, and later the thigh, hand, and arm become affected. This peroneal form, like the muscular dystrophies, is apt to occur in families. A marked peculiarity is the slow, halting onset. Years may elapse, for example, before the arms are affected. The disease is apt to be fairly symmetrical, though one leg may be attacked before the other. There are sometimes slight numbness and pain, but no objective anæsthesia. The disease begins in

early life, usually before twenty. Club-foot is common. The prognosis is better than in the arm type, but cure cannot be expected.

It would seem that our case conforms fairly well to this description, excepting that there is no family history; I am therefore inclined to favor this diagnosis. It only remains to be seen whether the upper extremities become affected later.

The pathology of this disease is not yet established. Some authorities favor primary degeneration of the nerves (parenchymatous neuritis); others, degeneration of the anterior horn cells. If either locality must be chosen for the primary seat of the lesion, I should be inclined to agree with the latter view. Very likely, however, the entire motor neuron suffers, from the cell to the periphery.

Treatment will avail little for this patient, but it seems not impossible that something may be accomplished in an orthopædic way, and I shall recommend her consulting Dr. Goldthwait, who has busied himself extensively of late with tendon-transplantation. This is not a new operation, Nicoladoni having already in 1881 attached a peroneal tendon to the tendo Achillis, and a few other surgeons have reported similar operations. The subject had not, however, received the attention it deserved till the appearance of Goldthwait's paper in 1896. This operator reports a large number of successful cases. I do not mean successful as regards curing the paralysis, but in the way of improving the position of the foot and facilitating locomotion.

The method, in brief, is something like this: In case the gastrocnemius is wasted but the peronei unaffected, the tendon of the intact peroneus longus (an extensor) is cut and attached to the tendo Achillis. In case of paralysis of the tibialis anticus, as in this case, the peroneus tertius (a flexor), if intact, may furnish a tendon for insertion into that of the paralyzed muscle. Unfortunately, in this case the peroneal group is affected. The next question presenting itself is, then, the advisability of transplanting one of the tendons passing behind the inner malleolus,—for example, the flexor longus pollicis, the flexor communis digitorum, or the tibialis posticus.

Dr. Goldthwait tells me that he has performed this variety of the operation with improvement in the usefulness of the foot, though the results are not so satisfactory as when the flexor muscles are made to do flexor work.

## SPECIFIC BASAL MENINGITIS.

This young woman presents several points of interest. A superficial examination would place her in quite the wrong category and deprive her of the opportunity for proper treatment.

She has been married a little over a year, and her history prior to a week ago is negative. At that time she noticed deviation of the face to the right side, coming on quite rapidly, with no other symptoms beyond moderate left-sided temporal pain. The facial paralysis is apparent; she cannot wrinkle the left side of the forehead, cannot close the left eye, cannot raise the left wing of the nose, or pucker the lips as if to whistle. In smiling and showing the teeth, the face is drawn to the right. This combination of symptoms corresponds perfectly to the familiar form of peripheral facial paralysis known as "Bell's paralysis." In this disease the nerve is invaded after entering the skull, either by aural disease or by inflammatory products resulting from neuritis (so-called rheumatic), the thickened sheath compressing the nerve in the Fallopian canal.

The fact that the whole face is affected, including the forehead, precludes disease at the cortex of the brain or in the internal capsule.

It must be remembered, however, that disease of the nerve at the base of the brain may produce a paralysis identical with that in the Fallopian canal. It is always wise, therefore, to look into the functions of the other cranial nerves before diagnosticating simple peripheral paralysis. The wisdom of the precaution is evident in this case, for, on asking her to move the eyes up and down and sidewise, we find that the left eye does not follow completely on outward movement; in other words, we find paralysis of the abducens nerve on the left.

Testing the sensation of the face (trifacial nerve), we find that a light touch is not felt on the left side. Let us mark out the boundary of this numbness, to see if it conforms to any special nerve-supply. The speediest method is to start in a region assuredly numb, and proceed in radiating lines in all directions till points of normal sensation are found. A line connecting these points represents the boundary of the anæsthetic area. I tell her to close the eyes, touch her lightly with the end of a piece of paper on the side of the nose, and ask her to tell me when she feels it. She makes no response. I proceed towards the mędian line, and as soon as I reach it she states that the

touch is felt; proceeding upward by successive touches I find that she feels nothing until I reach well into the hair; proceeding downward the limit of numbness is reached at about the edge of the chin; proceeding out over the cheek and temple in lines perhaps half an inch apart, and marking the limit of numbness in each line, then connecting these points, we have as a result this definite boundary.

FIG. 3.

Referring to the chart (Jakob and Strümpel),[1] we find that the line exactly represents the area supplied by the three divisions of the fifth nerve, excepting the branch of the auriculo-temporal, which supplies the temple over the zygoma. This result is sufficiently accurate to assure us of the involvement of the fifth nerve, excepting the branch just mentioned.

We must look, then, for a point at which the seventh, sixth, and fifth nerves converge. The only spot answering this demand is the base of the brain.

Having established the *seat* of the lesion by the objective *signs*, we seek to determine its *nature*, in which quest the principal aid is the *history*.

The onset was probably too rapid for a tumor, especially in the absence of preceding headache, vomiting, dizziness, mental defect,

[1] Certain authorities represent the cheek as supplied by the auricularis magnus, from the cervical plexus, but this very case tends to confirm the charts of, *e.g.*, Hasse, Jakob and Strümpel, and Dana, who assign the cheek to the fifth nerve. There is certainly no reason here to suspect disease of the cervical plexus.

or other general symptoms of new growth. No source of abscess is found, there is no special malaise, no change of temperature, no chill or other sign of this serious lesion.

We are brought, then, by exclusion, to basal meningitis, and, in view of the frequency of the specific form (gummous meningitis, commonly known as specific disease of the base), we are led to make further inquiries into the history. It seems that about ten months ago she had an eruption in the skin, followed by loss of hair.

We are justified, therefore, in regarding this the probable pathology, and shall put her at once upon the appropriate treatment.

It is rare for this disease to attack the brain within a year of the initial lesion, but such cases are not unknown. I remember only one other case coming under my own observation in which the period was less than twelve months.

The escape of the region ordinarily supplied by the temporal branch of the auriculo-temporal nerve I am unable to explain.

# POLIOMYELITIS AND ARTHRITIC MUSCULAR ATROPHY AFFECTING THE MUSCLES OF THE RIGHT ARM AND SHOULDER.

CLINICAL LECTURE DELIVERED AT THE ARAPAHOE COUNTY HOSPITAL.

BY J. T. ESKRIDGE, M.D.,
Denver, Colorado.

GENTLEMEN,—The case that I wish to study with you to-day is one of unusual interest, both in regard to diagnosis and prognosis.

The patient, William N., aged forty-seven years, single, white, a miner by occupation, was born in New Hampshire, but has lived in Colorado during the past seven years. Further than that one sister died dropsical, the family history, so far as he knows, is negative.

So far as he remembers, his first illness was in 1885, when he suffered from an attack of facial erysipelas. In November, 1889, he contracted pneumonia, and during the year 1890 he had erysipelas of the face a second time. From the last date he remained well until May, 1896, when he was injured in a mine. He was working in an upright shaft and a bucket of ore was being hoisted above him. When the bucket was about one hundred and twenty-five feet above his head, it tipped, and some pieces of ore weighing several pounds fell down the shaft and struck him on the head and right shoulder. As the ore fell it rebounded from one side of the shaft to the other and thus had its momentum greatly lessened, else the patient would not be living to-day to tell his story. However, his scalp was cut just above the occipital protuberance, his right shoulder was bruised, he was knocked down, but he does not think that he lost consciousness, although he felt dazed. He was able soon after the injury to climb the ladder to the top of the shaft and seek a physician, who dressed his wounds. He remained in bed most of the time for the next two weeks. His head and shoulder were very painful. At the end of that time, on leaving his room, he contracted pneumonia, probably

as a result of his depressed physical condition. By the middle of June, 1896, he thought that he had about recovered from the attack of pneumonia, but five days later he had a relapse of his lung trouble and was much prostrated. Phlyctenular ulcers appeared on the corner of the right eye, and he also began to complain of numb and tingling sensations from the shoulder to the elbow in the right arm. He did not rally well, and on July 12, 1896, he was admitted into the St. Luke's Hospital of Denver under the care of Dr. Bonney, one of the visiting physicians to the hospital. Dr. Bonney found an unresolved pneumonic process involving the lower portion of the right lung. On account of the paralysis of the right arm, he asked me to see the patient in consultation with him, and, a few days later, turned him over to me.

*Examination, July 13, 1896.*—The patient is greatly emaciated, weak, and anæmic. The temperature is nearly normal in the early part of the day, but rises to 100° or 101° F. during the early evening; pulse is weak and varies from 90 to 100; respiration is regular, but is 30 per minute. The lower half of the right lung is partially consolidated and filled with bronchial, subcrepitant, and crepitant râles. The bronchial râles are quite moist and expectoration is profuse. The sputum presents the appearance of muco-pus slightly tinged with blood. The cornea of the right eye is cloudy from recent phlyctenular ulcers.

He complains of pain in the right shoulder, and of a dull, heavy pain and numb and tingling sensations in the right arm and extending down to the finger-tips. On carefully testing him, it is found that all forms of objective sensory phenomena are normal in the right arm and shoulder. The deltoid, the infraspinatus and supraspinatus, the upper portion of the pectoralis major, the biceps, and the triceps muscles are greatly wasted. The deltoid and biceps muscles are completely paralyzed. The other affected muscles still retain some power, but are very weak.

On account of his nervous and prostrated condition, a thorough electrical test was not made of the wasted and paralyzed muscles. The most affected muscles, however, failed to respond to the faradic current.

Because of his limited means, the patient remained in the hospital only two weeks. During this time his lung trouble nearly cleared up, he gained considerable flesh, and felt much better. The biceps

muscle regained sufficient power to enable the patient to flex the arm at the elbow, a movement that he could not accomplish two weeks previously.

He was admitted into the nervous ward of the Arapahoe County Hospital, October 20, 1896. I made a careful examination of his condition three days later.

His lungs appear to be in nearly a normal condition; so are the heart and abdominal viscera.

There is no ataxia; his gait is perfect and the muscles of the legs are strong and well developed. Dyn. R. 84; L. 134.

*Right Arm and Shoulder.*—The forearm and hand muscles are fairly strong and are not much wasted.

The whole of the deltoid is atrophied, but the middle portion is more affected than the anterior or posterior part. The supraspinatus is quite small in comparison with its fellow of the opposite shoulder. The lower portion of the trapezius, the upper portion of the pectoralis magnus, and the serratus magnus are smaller than the corresponding muscles of the other side. The triceps and biceps muscles are atrophied, the latter to a greater degree than the former.

When I ask him to abduct his right arm, you observe that he is unable to raise the arm beyond a point about midway between the body and the horizontal position. If I attempt forcibly to abduct the arm so that it is at right angles with the body, he complains of great pain in the shoulder-joint and in the region of distribution of the circumflex nerve, so that voluntary movements of the arm are not only limited, but forced movements are also restricted on account of partial ankylosis of the shoulder-joint.

The deltoid muscle is almost completely paralyzed, and the lower and middle portions of the trapezius, the levator anguli scapulæ, the supraspinatus, the serratus magnus, the triceps, and the biceps are exceedingly weak. The fibres of the upper or clavicular portion of the pectoralis major that act in association with the deltoid in abducting the arm and raising it to a horizontal position are paralyzed.

Before calling your attention to the diagnostic significance of paralysis of a portion of a muscle that acts in association with another muscle that is paralyzed, and the power of this part of the muscle to contract when it acts in association with other muscular fibres that are not paralyzed, I wish to demonstrate to you the multiple action of the pectoralis major muscle on the normal side and the loss of its

associated action with the paralyzed deltoid on the right or affected side. If I raise the patient's left arm directly in front of him and hold it at right angles with the body, then request him to endeavor to depress the arm, the lower or costal portion of the pectoralis major and the latissimus dorsi muscles contract and would adduct the arm if it were not for the resistance that I interpose. While the arm is in the first position, if I now slightly depress it and request the patient to try to elevate it against my efforts to hold it in a depressed position, the deltoid and the clavicular or upper portion of the pectoralis major muscle contract. Again, if I elevate the arm to the horizontal position while I still hold it directly in front of him, and request him to bring the arm directly across the chest towards the opposite side of the body, the upper and lower portions of the pectoralis major muscle contract.

On now elevating the right or affected arm to the first position in which the left arm was placed, and requesting him to depress it, the latissimus dorsi and lower portion of the pectoralis major muscles contract, just as occurred on the left side. But if the right arm is depressed slightly from the horizontal position and he is requested to elevate it, neither the deltoid nor the upper portion of the pectoralis major muscle contracts. On again elevating the arm to the first position and requesting him to carry it across the chest towards the opposite side, both the upper and lower portions of the pectoralis major muscle contract quite vigorously. You observe, then, that the upper portion of the right pectoralis major muscle is paralyzed for the movement which occurs in association with the deltoid muscle, the latter muscle being paralyzed in this instance, but that this portion of the pectoral muscle contracts quite vigorously when movements are attempted that occur in association with the lower part of the muscle, in which the fibres are in a normal condition.

Continuing our examination of the case, let us test the reaction of the muscles to the faradic current. For this purpose I will use the secondary current of the large-size Flemming battery. The strength of the current is indicated by the number at which the magnet is tapped. These numbers run from one to four. The higher the number, the greater the strength of the current. These I will represent by the Roman numerals i, ii, iii, and iv. The strength of the current is slightly intensified by withdrawing a cylinder which surrounds an

iron rod. This is marked from one to six. These numbers I will represent by the Arabic numerals.

Deltoid: R. will not respond to the strongest current; L. ii.

Upper portion of the pectoralis major: R. ii; L. ii.

Trapezius (lower and middle portions) : R. iii; L. ii+2.

Supraspinatus: R. iii; L. ii.

Triceps: R. iii+3; L. ii.

Biceps: R. ii−2; L. i+4.

The patient complains of constant pain in the right shoulder and of pain at the lower angle of the scapula and in the region of distribution of the circumflex nerve when the arm is forcibly raised to or above a point at right angles to the chest. There are no points of tenderness over the shoulder or arm. All objective sensory phenomena are normal. All the reflexes are normal except those of the right triceps and biceps tendons. These are considerably increased over those of the left arm.

It will not be a waste of time if I tarry with you to study the mechanism of some of the movements of the arm at the shoulder, especially, in this case, those concerned in the abduction of the arm to the horizontal position and the elevation of it above the head. The arm is raised to the horizontal position mainly by the deltoid and clavicular portion of the great pectoral muscle. Abduction is slightly aided by the supraspinatus as it moves the arm forward and rotates it inward. After the arm has been raised to the horizontal position, these muscles can carry it no higher, and it is mainly carried above the head by the middle and lower portions of the trapezius and the serratus magnus from their action on the scapula and shoulder. The middle portion of the trapezius elevates the scapula and shoulder, while the lower part carries the scapula towards the spine and pulls the shoulder backward. The serratus magnus rotates the scapula on the inner angle and throws the acromion upward, but is opposed in this movement by the rhomboids and levator anguli scapulæ. The rhomboids and the levator anguli scapulæ, by fixing the scapula in different positions, aid the serratus magnus and the trapezius in forced elevation of the arm, but the last-named muscle is able alone to carry the arm above the horizontal position. This unaided action of the trapezius is weak.

I wish to impress upon you the fact that movements and not muscles are represented in the cord and brain. There are probably

as many representative centres in the cord for a given muscle as there are different associated actions for that muscle. In studying the function of the great pectoral muscle we found that the clavicular portion had three movements. The first is its action in association with the deltoid in abducting the arm and raising it to the horizontal position, the second depressing the arm in association with the latissimus dorsi and the third bringing the arm in front of the chest, aided by the costal portion of the pectoralis major muscle. In the cord we find that the great pectoral muscle is innerved by cells from three segments, the fifth, sixth, and seventh cervical. The deltoid has three associated movements, but two of them—the first bringing the arm across the chest, the second abducting the arm to the horizontal position—are performed in association with groups of muscular fibres that act together with the clavicular portion of the great pectoral muscle and the supraspinatus. The deltoid is innerved by cells from only two segments of the cord,—the fourth and fifth cervical. The supraspinatus muscle has, so far as I can now recall, only one associated action, slight abduction of the arm, and is innerved by cells from only one segment,—the fourth cervical. This muscle in health acts in association with the deltoid and clavicular fibres of the great pectoral in performing only one movement. When the deltoid is paralyzed, the supraspinatus is able to produce slight abduction of the arm. It apparently has only this one function.

From these studies of the different movements of the arm and shoulder we may draw the following conclusions: 1. When groups of muscles that act in association with one another are paralyzed, the lesion producing the paralysis is probably not in the nerves or muscles, but more likely is in the cord or brain. 2. When a single muscle, or when muscles that do not contract in association with one another, are paralyzed, the lesion is probably either in the nerves or in the muscles themselves.

To Dr. Beevor, of England, is due the credit of calling attention to the diagnostic value of studying the associated action of muscles.

*Diagnosis.*—In arriving at a diagnosis in the study of a case of the kind that is before us, we must bear in mind that one or more of several causes may produce symptoms somewhat similar to those that we have been studying to-day. We must consider the possibility of neuritis, injury of the muscles, progressive muscular atrophy, a focal lesion of the brain or cord, and, finally, arthritic muscular atrophy.

We have seen, from a study of the associated action of the muscles in this case, that the lesion, in all probability, is not in the nerves or muscles. A neuritis would be further excluded by the preservation of all sensory phenomena of the affected parts and by the absence of sensitive points or of pain in the course of the nerves.

Traumatism of the muscles would not be attended with great muscular wasting, profound electrical changes, nor the involvement of muscles that act in association with one another. On the other hand, there would be a history of an inflammatory condition of the muscles, and they would have recovered entirely before this time, or else have remained in a state of chronic inflammation and be more or less painful to manipulation.

Progressive muscular atrophy of myopathic origin does not come on acutely; it begins earlier in life, is usually hereditary, affects other muscles than those we find involved in this patient, and is not attended by the pronounced electrical changes present in this case.

*Focal Lesion of the Brain.*—An irritative cortical lesion of the brain is often attended by great wasting of the affected muscles. It is possible for the shoulder muscles to be involved and the forearm and hand muscles to escape if the lesion is limited to the cortical centre that controls the shoulder and upper arm movements. Practically, however, in all cases of marked muscular wasting due to irritation of the cortex of the brain that have come under my care, the muscles of the distal portion of the limb have been more affected than the proximal ones. The involvement of muscles acting in association with one another occurs from lesions of the brain cortex. Traumatism to the head lends aid to the suspicion of a brain lesion. The marked electrical changes found in this case, however, would not occur from a brain lesion.

*Focal Lesion of the Cord.*—The only lesion of the cord that would produce marked muscular wasting and electrical changes, without affecting sensation, is a focal poliomyelitis. Could all the symptoms present in this case result from poliomyelitis? The involvement of muscles that act in association with one another for the performance of given movements and pronounced muscular wasting with marked electrical changes are the usual symptoms of a lesion in the anterior horns of the cord. In addition to these, in the adult, rheumatoid pains are common in the affected limbs and the paralyzed muscles are not infrequently tender and painful. The only symptom presented

by this case that does not sometimes occur from poliomyelitis is ankylosis of the shoulder-joint.

*Arthritic Muscular Atrophy.*—If all the symptoms except those of the joint trouble may be accounted for from poliomyelitis, why is it necessary to consider the arthritis further than as a complication without its having any relation to the muscular wasting? In the first place, most, if not all, cases of inflamed and painful joints are attended by muscular wasting of the extensor muscles of the affected joint, and in these cases movement of the limb at the injured point causes pain to radiate through the nerves that supply the muscle affected by the joint trouble. This patient complains of pain in the nerve (circumflex) that supplies the deltoid, the muscles usually involved in injuries of the shoulder-joint.

In the second place, the prognosis and treatment are modified by the joint trouble.

In cases of pure arthritic muscular atrophy, the electrical reaction of the affected muscles to the faradic current is nearly normal, the reflexes are increased, and only the extensor muscles of the limb at the affected joint are involved, as a rule.

*Prognosis.*—If the marked atrophy of the deltoid muscle seen in this case was entirely due to poliomyelitis with loss of faradic irritability, at the end of six months from the beginning of the disease, little hope could be entertained of much improvement in this muscle. On the other hand, the atrophy that is due to the joint trouble will disappear if the latter can be cured. While I am guarded in the prognosis and tell him that all the muscular weakness and wasting will not be gotten rid of, yet I feel like encouraging him to look forward to obtaining fair use of the joint and considerable improvement in the muscular weakness. We must remember that a partial recovery in cases of this kind requires many months, or a year or more.

*Treatment.*—Little can be done directly for the cord lesion at this late date, but indirectly something may be accomplished in this direction, as well as in benefiting the affected muscles, by improving the patient's nutrition by means of iron, quinine, strychnine, arsenic, and good food.

The weakened and wasted muscles must receive especial attention for a number of months. All the muscles except the deltoid respond to the faradic current, and for these this current should be employed every alternate date. It should be only strong enough to

produce a mild contraction of the muscles: a stronger current is likely to exhaust. For the deltoid, a weak galvanic current will be necessary. Massage every day, or alternate day, will stimulate the muscles and improve their condition.

It is useless to treat a muscle wasted from arthritis unless attention is paid to the diseased joint. At the same time that the nutrition of the muscles is receiving attention, the adhesions of the joint must be broken up and the resulting inflammation combated by appropriate measures.

# Surgery.

## SPINA BIFIDA; SUB-BICIPITAL HYGROMA; DOUBLE HARELIP WITH PROJECTING INTERMAXILLARY BONE.

CLINICAL LECTURE DELIVERED IN THE UNIVERSITY CLINICS, BERLIN.[1]

BY PROFESSOR ERNST VON BERGMAN,

Director of the University Surgical Clinic and Professor of Surgery at the University of Berlin, Germany.

GENTLEMEN,—Let me first show you to-day the cases operated upon eight days ago. The first was a child, two years of age, who, you will remember, had a rapidly growing tumor of the testis. We decided that it would be one of two things: a sarcoma, which is not impossible even at this early age, or a dermoid cyst, though it was more likely to be the latter. In either case operation would be indicated with a tumor growing as rapidly as this. On extirpation it proved to be a dermoid.

You know the tendency of these tumors to contain tissue elements from various parts of the body. Epithelial elements and structures that are modifications of them, such as teeth and hair, are the most common; but portions of any kind of bodily structure may be found. Here, under this microscope, you will see that in this tumor there were a number of characteristic brain cells,—the large spindle and irregularly polygon-shaped cells, with their many processes, which occur in the brain cortex. The etiological factor in a case of this kind is evidently embryonal inclusion, though the varied structures that occur in these tumors sometimes might make us think of a species of parasitism.

The next case was a radical operation for hernia by Kocher's

[1] Reported by James J. Walsh, Ph.D., M.D.

method. As I told you a week ago, ordinarily I prefer Bassini's method for the radical cure of hernia, but where the hernia is not a large one, and there are no complications in the shape of adhesions of the sac, I think that Kocher's method may be used with excellent satisfaction. It takes but a short time, does not expose the patient very long to the risks of anæsthesia or to the shock of operative manipulations, and as tissues are not much interfered with vital resistance is not lowered and there is very little danger of infection. The results it gives are excellent. While there is not much danger of infection, there is some danger of mortification ensuing. The manipulation of the sac in getting it into the original opening in the canal, and then out through the new opening made for it in the aponeurosis, may lead to a twisting of the sac on its axis and so interfere with its blood supply. Gangrene thus resulting is one of the surprises the surgeon sometimes meets on taking off his dressings for the first time. Here everything is in good order, and after a few days more the patient will be allowed to sit up for a while each day.

The next case is the one in which four weeks ago we did a high operation for a large vesical calculus. As there had been a temperature of 40° C. for some days preceding the operation, which seemed to be due to an infectious cystitis, we left the wound open for the sake of thorough drainage, and the temperature promptly declined. The wound was left open for two weeks and then allowed to close. The patient is now in a condition to go home, though of course there is need of some care as yet to prevent a recurrence of his cystitis. The vesical mucous membrane still remains delicate, but with normal resistant power. His urine will have to be kept thoroughly alkaline for some time, and any abuse of alcoholic liquors or a too exclusively animal diet will have to be guarded against. He will be advised, too, to drink plentifully of water and to keep his bladder regularly evacuated.

Our first operative case for to-day is the child that has just been brought in. It presents, as you see, a tumor in the sacral region. This tumor is covered with a smooth, bluish, somewhat translucent membrane, which resembles to some extent the membrane covering the umbilical cord (navel string) in the fœtus. The tumor is cystic in nature, elastic to the touch, evidently containing fluid communicating with some deeper spaces, since even on gentle pressure its tension may be reduced some.

We have been very careful in these manipulations, as you see, because the situation of the tumor in this region just over the lower end of the vertebral column is characteristic for that congenital anomaly known as spina bifida, with the consequent tumors due to failure of the embryonic neural arches to properly close in this region. As these tumors are often in connection with the central canal of the cord and through that with the ventricles of the brain, the necessity for careful handling in the examination of it is evident. Besides, such tumors often contain nerve fibres for the nerve trunks of the lower extremity, and these not infrequently have their course along the inner superficial surface of the tumor, so that careful manipulation is doubly needed.

The bluish appearance of the tumor, with its smooth delicate covering, and this reddish velvety appearance, which we discover on closer inspection on its upper surface, is characteristic for that form of congenital anomaly of the spinal cord which Recklinghausen has called myelomeningocele. The nervous, *i.e.*, myelic substance, as well as the meninges and the spinal membranes, share in the incomplete closure of the neural canal which is observed in this case. Here in the midst of the little groove, which occupies the centre of the red velvety patch, there is an opening into which we can pass a small probe. This is the central canal of the cord opening on to the surface practically, though there may be a valve-like action of the soft tissues usually keeping it closed.

The inner surface of this groove is adherent to the nervous substance of the cord itself. The puckered velvety appearance produced where this depression sinks in is due to the inner surface of the pia—the leptomeninges in general—which turns in here. The rest of the tumor, too, is covered by pia, though there has pushed out over it from the skin edges a thin transparent delicate layer of epithelial cells which has modified its appearance. Where the bony arch is absent the dura is also absent, since it acts as the periosteum for the bones of the spinal canal and so is intimately associated with them when anomalous defects occur.

Besides this myelocele, Recklinghausen recognizes two other forms of tumor in this region which bear intimate relations to that condition. They are the myelocystocele and the myelocystomeningocele. In these the same split in the neural arches occurs as in myelocele, but takes place very early in embryonal life. Later, the

central canal closes and in the myelomeningocele—that is, in the cavity formed by meningeal and nervous substance—fluid collects, the pia and skin with the underlying subcutaneous connective tissue form protective coverings over the part through the defect in the vertebræ, and the dura remains. The same thing holds for the myelocystocele except that here the pia is also defective and does not form one of the coverings.

In either case, the lack of a bony wall to protect the part from the internal pressure of the cerebro-spinal fluid leads to dilatation of the end of the spinal canal, with the production of the characteristic cystic tumors. The collection of fluid in such cases is within the substance of the cord. A connection between it and the central canal of the cord can be nearly always easily demonstrated. The fluid is contained in a space lined with cylindrical epithelium, and is often surrounded by a membrane-like thinned-out layer of nerve substance from the cord.

As to whether in some of these cystic forms of spina bifida there occurs one in which the dura is present, though not the bony wall of the vertebral canal, is as yet doubtful. Careful investigations of such cases in order to decide this important question are needed and will well reward the time and labor spent on them. In the form generally known as spinal meningocele, the fluid collects in the subarachnoid space, or, if the dura is really present, in the subdural space, and it is only the membranes which under the pressure of the cerebro-spinal fluid prolapse through the defect in the bony arches and form the tumor. Only occasionally in such cases does a strand of the spinal cord arch forward to the level of the bone split, or a little above it, to become part of the tumor. As when the collection of fluid is in the subdural space this prolapse of nerve tissues does not take place, it may easily be seen how much further investigation of this subject and the discovery of the characteristics of this simpler anomaly will lead to confidence in the treatment of such cases. It is in certain of these not well understood simpler forms of spina bifida that the various modes of treatment which have been suggested have been so readily successful, while in other cases they have had a fatal issue.

Usually the tumors in this region are covered, first with normal skin, then subcutaneous connective tissue, then the leptomeninges, and finally a thin layer of nervous substance, often defective here and there, and sometimes represented by a continuous layer of epi-

thelial cells lining the inner surface of the pia. This we can scarcely fail to recognize as the ordinary lining of the central canal, though of course for this recognition the microscope is absolutely necessary.

The tumor in this case is not very large and the defect in the vertebral canal not very extensive. The embryonic defect may affect the laminæ of most or even all of the vertebræ. The rachischisis (split of the spine) may be either a holorachischisis (complete ὅλος whole), or merorachischisis (partial μερὸς part). The spinal defect may be only a complication of a corresponding split in the skull,—kraniorachischisis. For, after all, the skull represents ontogenetically vertebral plates, which may still be recognized in the embryo, and which have taken on a specially purposive exaggerated development in the course of evolution.

These extensive defects, kraniorachischisis, and even complete or multivertebral rachischisis, are incompatible with life. It is wonderful, however, how much of the spinal column may be involved and yet the child be viable and live for some time after birth.

On the other hand, there is a pseudo-spina bifida, in which the opening in the spinal canal is very small, or has even become closed in the course of fœtal development, and nothing remains of the tumor but a hairy spot over the last lumbar and first sacral vertebra, or a lipomatous mass. These, of course, are not dangerous to life and demand no operative interference. They must, however, be carefully distinguished from spina bifida occulta, in which, notwithstanding the absence of the bony covering, there is no prominent tumor, or where, though the tumor is a lipoma, there is beneath it an opening into the spinal canal with all the dangers that the exposure of the central nervous system to external influences brings with it.

Ordinary spina bifida may be compatible with reasonably long life. A number of such patients have been known to reach middle age, and at least one case is reported where death took place after seventy. The condition is not so infrequent as is often thought and constitutes about one-sixth of all anomalies reported, occurring about once in 1000 births. The proportion of cases that outlive childhood is very small, however, even where nothing happens to set up acute inflammatory processes. The development of the nerves to the legs is often seriously hindered and paraplegia supervenes, partly from faulty development and partly from pressure on the nervous elements at this point.

In most cases we must think of operation then, and especially is this the case when we have the form of spina bifida that we have here,—a meningomyelocele. The groove here at the summit of the tumor and the sinus admitting a probe is in direct connection with the central canal. The slightest irritation and infection here, and it may occur only too easily from the exposed position of the tumor, will set up an infectious process in the cord itself and its membranes, and spinal meningitis will close the scene.

These cases, too, are least adapted for the palliative operations, the emptying of the sac and the injection with irritants to set up adhesive inflammation, which is so often successful when only the membranes are involved. The irritative inflammation here would affect nervous elements and defeat its purpose of protecting the cord by directly injuring it. In many of these cases the direct operative treatment is not so difficult.

The nervous fibres are seen shining through the outer membranes after incision and lying between two delicate membranes. The upper membrane may then be removed, and after emptying the tumor of its fluid the other membrane, with the nerve elements, may be dropped back into the cavity of the vertebræ and the defect covered by a plastic operation. The danger of the operation is not so much due to the immediate injury inflicted on the cord as to the readiness with which infection takes place, especially afterwards, as the children are so hard to keep perfectly clean.

I always operate with the child's head well down, so as to prevent a sudden decrease of intracranial pressure, by allowing the exit of much cerebrospinal fluid. This sudden decrease of pressure may lead to wide dilatation of the blood-vessels of the brain, to extreme slowing of the blood current through them, amounting almost to stagnation in certain peripheral terminal arteries, with consequent intense lowering of brain metabolism and symptoms of malnutrition of the delicate nerve cells, which makes itself known by convulsions.

We shall endeavor to cover the defect in the vertebræ here, with a periosteal flap, and shall turn a flap of aponeurosis and of muscular tissue over it, to support the periosteum and give a firm wall at this spot. After the operation the child will be left lying for some days with its head lower, in order to avoid fluctuations in cerebral intraventricular pressure. This position will be constantly maintained, and not even for feeding and cleaning will the infant be lifted.

The danger of infection is here especially from below,—from the fæces and urine. After putting on a close-fitting gauze dressing, so strapped with adhesive strips that it cannot work loose, we shall cover the lower edge of the dressing, especially here near the anal furrow, with a thick layer of collodion. Special care will be given to the cleanliness of the child, and the outer dressing of cotton and gauze that we put over all will be changed whenever it becomes in the slightest degree soiled.

Our next patient, a woman of thirty-three, comes complaining of this swelling just above her elbow. It is the source of a good deal of annoyance to her, makes her awkward at her work, is even painful at times, and makes work harder to do and her arm more easily tired. We find a fluctuating tumor here, just under the tendon of her biceps. Its position and its consistency tell us what it is. There is just under the bicipital tendon at this point a synovial bursa, to enable the muscle to work easier and with less friction. This, like other bursæ, sometimes becomes enlarged and superabundant fluid collects in it, forming the so-called hygroma.

The tumor exists in this case on both sides of the bicipital tendon, and we can render the part on either side of the tendon more tense, by pressing on the other part. Since it gives her so much inconvenience we shall eradicate it. It has been tapped several times, but the fluid has always collected again. Now, we shall endeavor to dissect its sac entirely away, or we shall extirpate a portion of it and by scraping its interior surface set up an adhesive inflammation in what is left that will obliterate the cavity of the bursa and so prevent any further collection of fluid.

Very often these subbicipital bursæ are connected with the elbow joint, so that we must be extremely careful of asepsis in the operation. If it is connected with the joint we shall try to use the canal of connection as a pedicle and tie off the sac. After making the incision of the skin, as you see, I dissect it back carefully without wounding the sac, because while the fluid remains in it its walls are easy of recognition, and so we can isolate from surrounding tissues easily. Here, after our dissection, we find the connection with the joint that I anticipated, and here we shall tie it off.

We shall have the arm dressed in the flexed position at an acute angle, in order to avoid all tension on the wound and the stitches. After eight or ten days we shall have it gradually more and more

extended in the dressings. We have no contractures to fear here and need only think of the most suitable position to free the wound from all irritative tension and have it heal rapidly.

These subbicipital hygromata are not so rare as to be curiosities, and yet are rare enough to make mistakes of diagnosis possible, because the condition is not thought of as occurring here. For the hygroma beneath the olecranon, which makes a tumor at the elbow (miners' elbow), corresponding to housemaids' knee, the enlargement of the prepatellar bursa, there is never any hesitancy in the diagnosis. To have seen one of these subbicipital hygromata is a valuable object lesson. It is to be remembered that certain bursæ higher up in the arm, as beneath the deltoid, or anomalously beneath almost any of the muscles of the upper arm, may be liable to this hygromatous condition.

Our last patient for to-day is a child, a little over two months old, with a double harelip. As is usual in these cases, there is also a projection of the portion of the upper jaw in the middle line. This projecting bone is the process of the intermaxillary bone which usually disappears by fusion with the two lateral plates of the superior maxillary at an early period of fœtal life, but which in cases like this is anomalously persistent. This intermaxillary bone is of special interest to us Germans, because it was the poet Goethe who first pointed out its occurrence in man.

It occurs in all the animals, but up to Goethe's time was supposed not to occur in man; in fact, it was thought to constitute the distinctive osseous attribute which differentiated the human skeleton from that of the brute. Camper, the discoverer of the facial angle of the skull and its significance in anthropology, had publicly written to this effect shortly before Goethe made his discovery, and the great Cuvier was of the same opinion. Goethe was led to the idea that there must exist in man at least remnants of an intermaxillary bone, by his intimate persuasion of the unity of nature and man's bodily affinity with the animals. By careful maceration he succeeded in demonstrating the presence of the intermaxillary bone in young fœtuses, and succeeded in making good his position against the prejudices of distinguished scientists.

It is not the only thing we owe to the great German poet in the field of anatomy, or rather osteology and embryology. For, shortly after his discovery of the intermaxillary bone, while walking on the

Lido at Venice one day, he picked up a dried macerated sheep's head from the sand, and there flashed on him the brilliant conception that the skull was but a continuation of the vertebral column, that it represented a certain number of vertebræ modified, as I have said while talking of our first case, purposively, so as to protect and inclose the terminal enlargement of the central nervous system which the stage of evolution of the animal demands. Need I say that Goethe was more proud of his scientific discoveries than of some of his great literary work, and that we can thoroughly sympathize with his very pardonable pride under the circumstances?

These defective conditions which we have so often in the neighborhood of the mouth are due to the failure of the maxillary processes to properly unite. Five of them meet here, the intermaxillary, the two lateral superior maxillary, and the two inferior maxillary plates. At times epiblastic structures dip down into the clefts that separate them in embryonic life and keep them from normal union. The most frequent anomaly is the failure of one superior maxillary to fuse with the intermaxillary and the other superior maxillary, causing the ordinary single harelip. When this failure to unite is bilateral, then we have double harelip, though the separation is usually more marked on one side than the other, as it is here on the left side.

Sometimes the defects extend back and involve the hard and soft palates. Sometimes, too, it is the superior and inferior maxillary processes alone that fail to unite, and the condition known as macrostoma, congenital transverse fissure of the cheek, results. This condition is usually accompanied by some defect or excrescences in the region of the ear. Of course, these are not the only defects that may occur. There may be splits along the alæ of the nose, or defects of parts of the nose in connection with the oral anomalies, or combinations of abnormalities, to almost any extent.

As to treatment, all are agreed that by a plastic operation a great deal of improvement, sometimes practically complete closure of the defect, may be secured. It is true that when even double harelip is present with prominent projection of the intermaxillary process, as here, a certain involution of the parts with natural correction of the deformity takes place in course of time and Nature unaided brings about great improvement. During my recent visit to Russia, I saw in Prince Oldenburg's hospital in St. Petersburg a series of photographs of a case of double harelip with projecting intermaxillary

bone, which had been left entirely to itself. The resultant improvement in the course of time was most striking. A plastic operation will, however, help Nature very much, if properly done; so that it is not only justified but indicated.

As to when it shall be done, all surgeons are not agreed. It should certainly be done before dentition, for the teeth grow more normally, and the irritation of teething is in itself an unfavorable circumstance for the rapid, easy healing after operation, and disturbs the general well-being, so as to make plastic surgery less successful. The operation should not be done too young, as very young infants do not stand loss of blood well, and sometimes in double harelip quite a little blood is lost. Besides, the very young infant's tissues are soft and yielding, and harelip pins easily tear through them, thus preventing proper apposition after operation. I have done the operation when parents insisted on very young children, but prefer to wait until the child is about the age of this one here, two and one-half months old.

I prefer to give no general anæsthetic. Local anæsthesia is secured by the injection of a small amount of a one-per-cent. solution of cocaine. The child is carefully wrapped up in sterile cotton, so as to prevent its taking cold during the operation, and keep it from hurting itself by its struggles. Over the cotton are wrapped reasonably firmly some gauze bandages. Then the child is fastened on an inclined plane at an angle of about sixty degrees, this being the position in which I think there is the least liability of its drawing into its trachea blood or oral contents.

The first part of the operation must consist of the disposal of the projecting intermaxillary bone. As on the presence of this will depend the proper support of the lip and the future incisor teeth, I prefer always to preserve it. I prefer to do the operation in two stages then: first, the pushing back of the intermaxillary bone into position; and, second, the bringing of the edges of the cleft in the lip together.

The first part of the reposition of the intermaxillary bone I shall do to-day, and then allow a week to elapse before I proceed with the harelip operation proper. I loosen the attachments of the bone for some distance back by means of a small chisel, carefully avoiding on each side the arteriæ incisivæ, which sometimes cause troublesome breathing that requires the actual cautery to stop it. While I have,

as you see, the actual cautery ready for immediate use, because the loss of even a little blood is important for so young a child, I hope not to have to use it, as the cauterization affects the blood circulation of the part seriously, and so interferes with the success of the plastic work. As to the success of my efforts at reposition of the intermaxillary, you will be able to judge next week.

# INTERESTING CASES OCCURRING IN THE SURGICAL CLINIC OF

PROFESSOR W. W. KEEN, M.D., LL.D.,
In the Jefferson Medical College Hospital, Philadelphia.

REPORTED BY A. G. ELLIS,
Medical Student

## SUBDURAL DRAINAGE OF THE LEFT VENTRICLE FOR HYDROCEPHALUS.

Gentlemen,—We have here a child, four months of age, suffering from acute hydrocephalus. The case was kindly referred to me by Professor E. E. Graham, and is of unusual interest, and an unusual operation will be done in an attempt to save its life. The child's head has enlarged very rapidly, the circumference increasing an inch in one week. Lumbar puncture was done, but without success. In a case of internal hydrocephalus such as this is, the fluid can sometimes be drawn from the ventricles into the spinal canal through the foramen of Magendie and evacuated by lumbar puncture. In the present case only two cubic centimetres could be obtained in this way, with the advantage of showing, however, that the fluid was sterile.

The operation to be done is the ingenious one suggested by Cheyne, of London,—*i.e.*, subdural drainage of the lateral ventricle. The ventricle is punctured, three or four strands of catgut are slipped in, and then, instead of bringing the ends out through the bone and scalp, they are simply pushed under the dura. By this means the fluid, it is hoped, will be drained into the subdural lymph-space and absorbed. The great advantage of this lies in the fact that there is no external wound to dress day after day, and therefore no danger of infection. I care not who the surgeon is or what his technique may be, if he dresses a wound in which drainage makes this necessary day after day, and it may be two or three times a day, it is only a question of time when infection will follow. By Cheyne's method the skull is shut up, and the wound in the brain must be aseptic.

After reflecting a flap of scalp, I make a small trephine opening a half-inch in diameter, and then I make a small opening in the dura. By using rather stiff catgut just taken out of alcohol, I can push the catgut through the thin cortex into the ventricle as soon as I have made the opening. A very little fluid flows out, nor do I wish a free escape at the time. If much fluid is taken out at once, convulsions follow, and death ensues on the spot or in a day or two. It is a question if this is not best in many such cases of unfortunates.

The other ends of the catgut are now slipped under the dura and the wound is closed. The stitches in the scalp are put in very close, to prevent leakage, as it is desirable to have the wound close up as soon as possible.

[Considerable leakage occurred during the first twenty-four hours. This was arrested by iodoform collodion. The child recovered from the operation, though the temperature immediately afterwards and for fourteen days rose as high as 105° and 106° F. No assignable cause for this could be discovered. The fever then ceased, but the child was not improved by the operation. The head, which in the month preceding the operation had increased one and one-eighth inches in circumference, at first shrunk, then again enlarged even beyond its former size. The child was still living several weeks after operation, but without any improvement.]

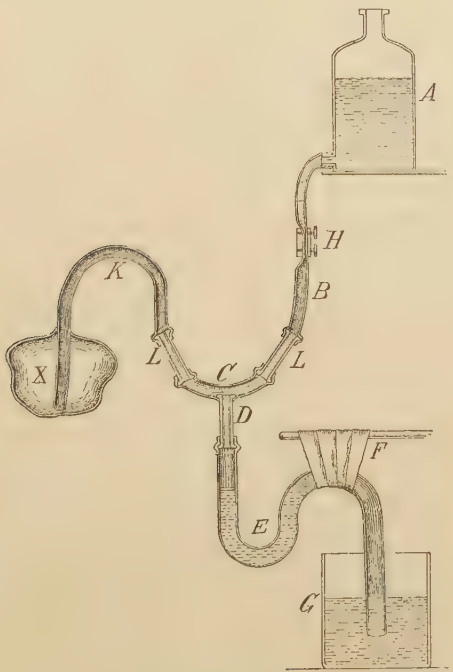
### A METHOD OF DRAINAGE AFTER SCHEDE'S OPERATION.

The next case to which I wish to call your attention is that of a man who was operated on last week. I bring him before you especially to show the method of drainage used since the operation.

This man had an old empyema, with a fistulous opening, which had existed for two years and a half. The opening was just below the second rib, higher up than I have ever seen before. The direction of the sinus and extent of the cavity could not be well determined by the probe. A T-shaped incision was made, the three flaps were drawn back, the opening into the thorax was found, and with bone-nippers I made an opening large enough to permit me to look in. Schede's operation as modified by myself was determined upon, and the second, third, and fourth ribs were removed, thus taking the whole chest wall, excepting the soft parts external to the ribs, but including the thickened plura, from the lower border of the first rib. The cavity was packed with iodoform gauze for three days.

Of course it was already an infected wound, and, not wanting the pus to collect or remain in the cavity, I used the plan of drainage introduced by Cathcart, of Edinburgh, and slightly modified by myself. A large jar of water is placed on a stand by the bedside. Leading from it is a rubber tube on which is placed a clip. This clip is not allowed to shut off the water entirely, but is held open with a small bit of tubing or other object so as to permit the water to run very slowly. This tube terminates in a "Y." This "Y" need not be of

FIG. 1.




Keen's improved method of drainage by siphonage.

glass: a good enough one can be made of tin, or even of two pieces of tubing united by end-to-side anastomosis. One of the upper tubes leading from the "Y" goes into the man's chest and is held in place by plaster, the other upper tube is connected with the bottle of water. The third or lower tube from the "Y" is fastened to the side of the bed in a loop so as to form a siphon. This, of course, must be lower than the one leading from the wound. There is also a piece of glass tubing inserted in the tube from the wound, so that the action of the apparatus can be observed. The tube forming the loop fills slowly

and then siphons over, thus making marked suction each time it empties. Only a slow trickle comes from the jar, so the suction is expended on the fluid in the cavity, which is thus drained out every few minutes.

[This man was desperately ill after the operation, but was rescued by hypodermocylsis. Unfortunately, this caused two sloughs as large as the hand on each thigh, but they finally healed. His highest temperature was 101.6° F. He is sitting up in bed occasionally now, in order to avoid bed-sores. In a day or two the stitches will be cut, to let the chest wall fall in.

An immense cavity was left by the operation, as the flap of soft tissues over the chest was far too small to cover its sides and floor. It is slowly filling up by granulation. Meantime the patient's general condition is excellent, and he is up and about. The microscopical examination by Professor Coplin suggests probably a pleural endothelioma as the origin of the empyema.]

### A CASE OF FOCAL EPILEPSY, WITH THREE FRACTURES OF THE SKULL, ONE OF THE BASE AND TWO OF THE VAULT.

This patient is a railroad brakeman from Florida, whose head was caught between a car and a piece of timber. He comes to us with three distinct lesions: First, there is a marked depressed fracture of the skull over the left temple, where an angular ridge is distinctly felt. Second, there is a small depression in the vault of the cranium over the right parietal bone, which was presumably a fracture. Third, at the time of the accident he had bleeding from the nose and also vomited blood, but had no bleeding from the ears. This would indicate a fracture of the base of the skull in the anterior fossa, and he has another lesion which confirms this diagnosis. The right optic nerve is atrophied and this eye is practically blind. This shows a lesion at the base of the brain in front of the optic chiasm. This is an irreparable loss for which nothing can be done. The fracture on the left temple is giving no trouble. But on November 1 the man had an attack of epilepsy, and four or five days after that he had a second attack. These started in the left foot, which began to shake before the attack came on. It is rather surprising that the arm is not affected also, but the leg centre seems to be the only one injured. Here is a case in which the knowledge of the localization of function assists the surgeon most markedly. Twenty years ago I should probably have trephined at the fracture on the left temple,

as that is so much more depressed and seems to have been so much more severe. But the history of his attacks clearly point to the much less marked depression on the right side, which is over the lower border of the centre for the left leg. The patient's epilepsy may be caused by laceration of the brain without fracture, or by a depressed fracture or by a fracture of the inner table only. On account of this uncertainty, the operation will be largely exploratory, but when the skull is opened whatever is necessary will be done.

I open the skull with a large gouge and hammer, as I shall not replace the bone and this is a quicker method than trephining. The dura is normal and the bone seems not to have been fractured, in spite of the depression. I will now apply a faradic current by a double electrode before opening the dura. I test the strength of the current first, and make it just strong enough to flex my index finger. Moving it over the centres of motion, we see the fingers of the left hand flexed and movement in the calf of the leg. There is no softness of the dura and no reasonable probability of a cyst or clot being present. But I will make a small opening in the dura, in order to be able to look in and see if the brain is normal. I shall make an opening small enough to be closed with one stitch. The advantage of such a small opening is that we are able to see if a larger one ought to be made, without making a large circular incision with perhaps no reason for it. I see that the brain looks perfectly normal, so the wound will be closed.

Hereafter the wound can be reopened and pieces of bone put in to fill the gap. You may wonder why nothing was done to the brain. It seems to me there could be no good in any operation on it. There is nothing to encourage interference with it. Even if an existing scar were cut out, the resulting one would probably be as bad, and in fact no scar is visible.

[Nine days after the operation he left the hospital, entirely well from the operation. His subsequent history is unknown.]

## TREATMENT OF EXSTROPHY OF THE BLADDER; THE RECTUM A SUCCESSFUL RECEPTACLE FOR URINE AND THE MENSTRUAL DISCHARGE FOR TWENTY-TWO YEARS AND SEVEN YEARS RESPECTIVELY IN TWO CASES.

This little baby has the rather unusual condition of exstrophy of the bladder, a malformation caused by failure of development in the anterior wall of the bladder and abdomen. The urine constantly

exudes and excoriates the skin, making a very deplorable state of affairs. Treatment, as a rule, is unsatisfactory. Various forms of flap operations have been devised, the best probably being the one in which a piece from above is turned down with the skin side towards the bladder and then a flap from each side is placed over this by sliding it sidewise. But this makes no urethra, and there is no possibility of making one. In these cases it is necessary for the patient constantly to wear a urinal. This, of course, is excessively difficult to keep clean and sweet. I have found that the best means of keeping such a urinal free from smell is to put a teaspoonful of chloral into it every time it is emptied. This answers the purpose well enough to make it a point worth knowing.

Recently another method of treating these cases has been devised by Fowler, and it is the method above all others in my estimation, though by no means free from danger. The operation consists first in opening the belly and finding the ureters. Then the rectum is opened and the ureters are implanted in its sides. This does admirably, and the patient is kept thoroughly dry except for the mucus secreted by the mucous membrane of the posterior wall of the bladder. But this is only slight in amount and can easily be attended to. I have recommended this operation in the present case when the child is old enough, and have advised the parents to wait some time. The child is so small now that the operation would be very difficult. When it is ten or twelve years old, or even before if it grows fast, the operation can be done.

The question naturally arises whether the urine will irritate the rectum, and if it will be retained under such circumstances. The rectum seems to answer such purpose very well. In confirmation of this, only last week I saw a patient with a most interesting history. She is a woman who during an attack of typhoid fever noticed that urine was escaping from the vagina. Soon after fæces also made their exit by this passage, the infection of the fever having caused gangrenous sloughs from both the recto-vaginal and vesico-vaginal septa. One of my colleagues and I did a number of operations to relieve this unfortunate woman, she being a social pariah on account of her condition. In all she had thirteen operations done. She was nearing the climacteric and was a widow. After the operation had been explained to her, she readily consented to have the vagina entirely closed. To obtain this result, at the last operation I excised

the remnant of the urethra left and totally closed the vagina. That was in 1876, and she has been well and dry and clean ever since and able to work as a nurse. Both urine and fæces now pass through the rectum, and while she menstruated that discharge also took the same course. The urine has never irritated the rectum, and she has done well ever since the operation, with the exception of one occasion, when a small calculus formed in the vagina and acted as a ball valve. It was readily crushed through the anus and the recto-vaginal fistula by a simple curved pair of hæmostatic forceps. She generally voids urine two or three times during the night.

I have recently had another case in which for an incurable vesico-vaginal fistula Professor Parvin seven years ago totally closed the vagina after making a recto-vaginal fistula. No trouble has been experienced from the urine and menstrual discharge in the rectum, but there has been such contraction of the recto-vaginal fistula as to give her great annoyance. Dilatation has relieved her entirely.

## A NOSE DEFORMED BY FRACTURE GREATLY IMPROVED.

Our next operative case is one in which an attempt will be made to restore the shape of a nose which has been deformed by a compound fracture. The patient is a girl who some time ago fell and sustained a compound fracture which has left a saddle-shaped nose.

I have had a dentist make a cast of this nose, and then from that a model by which he made a plate to be inserted. The plate is a double one, with an air space between in order to make it light. The plate is made of silver and then gilded. Along the margins is a row of holes, which have been left with the hope that granulation tissue will grow up through these and, becoming fibrous, will hold the plate firmly in place. The first operation of this kind by myself was done four years ago, and the gold piece is yet in place and has given no trouble. A similar case in Australia, done some years before my own, was doing perfectly well seven years after operation. Other supports have been used, such as celluloid, which does very well. Aluminum is too easily destroyed by the fluids of the tissues.

If there was no scar a small opening at the side would be made and the plate slipped in, but as there is a scar here, an incision in it will make it no worse. An incision is made, and the tissues are loosened from the bone and cartilage with a dissector, being very careful to avoid infection by not invading the cavity of the nose. A

few cases have given trouble by irritation, but this has not occurred in the two cases mentioned. The external wound will be sewed with a fine needle and the finest silkworm gut. The stitches will be removed as soon as possible. In order to guard against the possible collecting of blood under the plate, I shall put in a double strand of silkworm gut for drainage, which will be removed to-morrow. A dressing covering both eyes as well as the wound will be applied, great care being taken not to use too much pressure, as a slough might easily form over the plate.

[The patient made an excellent recovery by first intention. Some months later one corner of the plate, which was not pushed far enough from the wound, protruded and the plate was removed. A second attempt to insert the plate will be made in the autumn.]

## SARCOMA OF AN ECTOPIC TESTICLE.

We owe this case to the courtesy of Professor Hearn. The patient is thirty years of age, was married five years ago, but has no children. Six years ago the left testicle descended into the inguinal canal. Soon after the right came down the same distance. In this position they are subject to pressure and violence and are apt to undergo sarcomatous degeneration. At the present time there is a hard mass in the right inguinal region where the right testicle lodged. It is ten centimetres long and half as wide. The skin over it is freely movable, but the tumor is tightly adherent to the pelvic brim. There is another tumor, at least twenty centimetres in length and six in width, extending from the left of the umbilicus, under the liver and colon, towards the right side and the xiphoid. It is dense and also fixed. We have, therefore, (1) a partially descended testicle in the left inguinal canal, (2) a sarcoma of the right partially descended testicle, and (3) a large sarcoma in the retro-peritoneal tissue of the abdomen, in all probability secondary to the one in the groin. Operation is out of the question here. The man has come too late. An ectopic testicle should be placed in the scrotum by operation or, failing in this, be removed. It is generally of no value sexually, and this man would seem to belong to this class. I have seen one man who had both testicles in the scrotum, normally developed genitals, and normal sexual desire and its normal gratification, and yet who had not a single spermatozoön in the spermatic fluid.

In the last few years many operations for transposing an ectopic

testicle into the scrotum have been done, especially in France. Some years ago I assisted Professor J. Chalmers Da Costa in operating on such a case. He found that he could not bring the testicle down far enough, and therefore he dissected the cord loose from the posterior aspect of the testicle and simply turned the latter down into the scrotum, with an excellent result.

## MYXO-SARCOMA OF THE TONSIL.

This case has been an interesting one and has presented several serious phases. The patient is a girl eighteen years of age, sent to us by Dr. J. D. McLean. She presented herself several months ago with a growth in the right tonsil. An operation was urged at that time, but this was declined by her parents. The tumor had increased in size until it nearly filled the entire oro-pharynx and very seriously interfered with her breathing when she returned early in October. The glands of the neck were involved and they were first removed. My intention was to extirpate both carotids, as suggested by Dawbarn, to prevent recurrence, if possible, by starving the growth. By attacking the glands first and laying bare the vessels, I should have instant control of them in case of serious hæmorrhage when removing the tumor. While removing them dyspnœa became most alarming, and tracheotomy seemed imminent. To avoid this, if possible, about one-third of the tumor was cut off with a pair of scissors. One of the first steps in the operation on the neck had been to throw a loose ligature around the cartotid, so as to enable me instantly to control hæmorrhage should it be alarming. This was not required, as the bleeding was moderate. The relief was immediate, and the hæmorrhage was easily controlled by iodoform gauze. The Trendelenburg posture was of the greatest assistance. In fact, without it operation on the immense growth would have been almost impossible. After removing the glands in the neck, the external carotid artery was dissected out, its branches were ligated, and about four and a half centimetres of the artery excised. The remainder of the tumor was then removed through the mouth, without splitting the cheek.

When the patient had recovered, which she did nicely, Professor J. Chalmers Da Costa operated upon the glands and dissected out the external carotid artery on the opposite side. This operation proved a serious one. The glands were involved more than was suspected and were very adherent. So strong was the adherence that the jugular

vein was torn in removing them, but was secured by double ligatures. Then the external carotid was looked for, but could not be found. A dissection was made far up the neck without result, and then the carotid artery was followed down as far as possible, when it was found that there was no division on that side and consequently no external carotid to excise. This is a rare condition and made the operation a puzzling one.

[The patient recovered from the second operation. Unfortunately, two months after the operations there was a slight recurrence on the postero-lateral wall of the right pharynx, for which another operation will soon be done.]

### RECURRENT CARCINOMA.

This woman had her breast removed by another surgeon a year or two ago, and now presents the following condition. The right arm is œdematous to such a degree that it is at least twice as large as the left. The glands above the clavicle are involved and can be easily felt. No enlarged glands are to be detected in the axilla, but there must be involvement of those near the vein, as is shown by the œdema of the arm. The sternum is tender to the touch.

In the way of local treatment nothing can be done in this case. Only one operation is possible, and that is not very promising,—the removal of both ovaries. This treatment is in the experimental stage at present. Just what connection there is between these organs and the growth or hinderance of cancer is not known: whatever is said about it is only theory. It is thought that there is probably an ovarian secretion, which when poured into the system is favorable to the growth of cancer. The operation has been done several times. A very few of the patients have improved, but the majority go on to death. However, considering the comparative safety of abdominal operations, in a woman to whom the ovaries are no longer of functional value the procedure is worth trying. It is the only hope, and I shall suggest to this woman such an operation at an early date. [The operation was declined.]

### LEONTIASIS OSSIUM.

The last case is that of a girl ten years of age. Six years ago the left eye became inflamed and gave considerable trouble. Shortly afterwards a swelling was noticed in the frontal region above that eye. This enlarged, the eyeball being pushed downward, and optic

FIG. 2.—The case of leontiasis ossium after the second operation.

neuritis was present. A tumor of the frontal lobe was thought possible, although the absence of cerebral symptoms gave rise to uncertainty. There had been no mental symptoms, as is generally the case in frontal tumors. The child remained as bright as any one of her age would be. There was no epilepsy nor headache.

A large flap of skin was turned up, but on trephining the skull was found to be enormously thickened. The bone was so soft that it was easily gnawed away with the bone-forceps, and could even be shaved off with a scalpel. The dura was exposed over a large area. An examination of the orbit showed no necessity for interfering with its roof, as was feared. The bone was at least three centimetres thick. The fragments being diseased, it was not desirable to replace them.

[The child made an excellent recovery, and two months later a piece of celluloid was inserted to protect the brain. This operation also was followed by primary union.]

# VARICOCELE AND ITS TREATMENT; FIBROMA OF THE MALE URETHRA; RECTAL FISTULA.

CLINICAL LECTURE DELIVERED AT THE NORTHWESTERN UNIVERSITY MEDICAL SCHOOL.

BY A. E. HALSTEAD, M.D.,

Professor of Anatomy and Associate Professor of Surgery and Clinical Surgery in the Northwestern University Medical School; Professor of Rectal Surgery in the Chicago Policlinic; Attending Surgeon to the Cook County Hospital.

GENTLEMEN,—The first patient whom I shall present to you to-day is a young man who complains of having a swelling in the left side of the scrotum. His history is as follows: E. D., age twenty-three; occupation, waiter. Family history negative. He denies ever having had venereal disease of any kind. He first noticed about five years ago that the left side of his scrotum was somewhat larger than the right. About this time he became aware of an uncomfortable sense of fulness and some pain in the left testicle. This distress has gradually increased, the pain becoming greater and the swelling augmented. For the last two years he has suffered considerably from a dragging pain in the back, which is markedly aggravated in hot weather. The continuance of this pain has been a constant source of concern to him and has caused him to become despondent. His sleep is broken and a condition of extreme nervousness has resulted.

As the patient stands before you, it is plain that the left testicle is larger than the right. Palpation of these organs also demonstrates the difference in the volume of the two sides and shows the left testicle to be soft and almost painless. The veins of the cord are greatly dilated, and suggest to the touch, as is said, a bunch of earthworms. When we place the patient in a reclining posture, as you see, the size of the veins of the cord quickly diminishes and on slight pressure the testicle becomes perceptibly smaller. We can safely say that the condition we have here is a dilatation of the veins of the pampiniform plexus which receives the blood from the testicle and cord. This varicose condition of these veins is known as varicocele.

Varicocele usually develops about the age of puberty or shortly after. It rarely begins in childhood and seldom commences after middle age. When once a varicocele is well established, spontaneous disappearance practically never occurs.

The frequency of varicocele is variously stated,—from 2 per cent. by Sistach to 66 per cent. by Carré. Recently Senn found that in 9825 volunteers for the Spanish-American war 2078, or 21.17 per cent., had varicocele of some degree. We can safely assert that one out of every five men has some dilatation of the veins of the cord. In many of these cases the patient is not aware of this condition until his attention is directed to it.

The subjective symptoms of varicocele differ widely in individual cases. In some a very great dilatation of the veins causes no inconvenience. In other cases a small varicocele will cause great discomfort and at times affect the general health of the patient. In most cases the pain is referred to the testicle and cord on the side affected. In many the pain radiates from the testicle down the thigh and into the perineum. Frequently pain in the back is the chief cause of complaint. The pain is always aggravated by prolonged walking or standing or by any sexual excitement. When these symptoms have existed for some time, mental disturbance, such as anxiety and despondency concerning the ability properly to perform the sexual act, is a frequent sequence. In the so-called acute varicocele the pain and swelling may be so great that the patient is obliged to remain in bed for several days or weeks. These cases of acute varicocele are the immediate result of trauma and are very rare. In the great majority of cases the development of varicocele is extremely slow.

In the etiology of varicocele a number of factors play a more or less important *rôle*. Billroth attributed many to an inherited diathesis in which the veins seemed to be congenitally frail, either from a defect in the vessel wall or from a deficiency of valves. This is borne out by the frequency with which several members of the same family are found to have varicosities of the veins in some part of the body. Again, we frequently find varicocele associated with varicose veins of the extremities or with hemorrhoids in the same individual. Contusions of the testicle and cord are frequently followed by varicocele, either immediately or some time afterwards. Violent muscular effort, especially where the abdominal muscles are frequently called into action, as in lifting or carrying heavy weights, often acts as a cause of

varicocele. In general, any condition that will interfere with the return flow of blood from the testicle and cord will ultimately destroy the integrity of the valves of the veins and act as a determining cause. In this connection may be mentioned tumors of the abdomen, especially of the kidneys, and hernia.

It has long been noted that varicocele occurred with much greater frequency on the left side than on the right. Various reasons have been given in explanation of the great preponderance of left-sided varicocele. Lenoir and Sistach thought that the left abdominal muscles, particularly in right-handed persons, were more frequently contracted than the right, and therefore the circulation through the veins of the left cord would be interfered with more than that of the right side. The fact that the left spermatic vein joins the left renal at about a right angle, while the right empties into the inferior cava at a very acute angle, is frequently invoked as an explanation of the frequency of left-sided varicocele. The greater length of the left cord also predisposes to varicosities of these veins. Petit considers that the close proximity of the sigmoid colon to the spermatic vein influences the circulation of blood through this vessel and acts as a cause of varicocele. All of these factors undoubtedly have some influence in causing left-sided disease. The most important is, in my estimation, the peculiar relation of the left spermatic to the left renal vein and the greater length of the left cord and plexus.

The pathological changes which take place in the veins in varicocele are about the same as those found in varicose veins of the lower extremities. They are thickening of the vessel wall and a chronic periphlebitis with an increase in the connective tissue around the veins. In varicocele aneurismal dilatations or the formation of a connective-tissue tumor as the result of thrombo-phlebitis seldom take place, although they are common in varicose veins of other parts of the body. Occasionally the wall of the vein is greatly thickened and its lumen obliterated. As a result of the constant engorgement of the testicle with venous blood, the gland tissue begins to atrophy early. This atrophy is not apparent until the gland has been emptied of its blood. It is well in every case where an operation is to be considered to inform the patient of the probable atrophy of the organ before the operation is undertaken, so that the subsequent shrinkage of the testicle will not be attributed to the operation.

The treatment of varicocele may be either palliative or curative.

The former consists in applying a properly fitting suspensory and in generally improving the patient's mental and physical condition. He should be directed to keep the bowels regular and to avoid excessive fatigue and any sexual excitement. Standing for a long time, especially in hot weather, should be avoided. Cold douching of the scrotum is of benefit, especially in the early stages of the disease. Palliative treatment is indicated only in the mild form of the disease or in the early stages before pain is a prominent symptom. In large varicocele, even though not painful, atrophy of the testicle will surely result if curative treatment is not resorted to. In every varicocele where pain is a prominent symptom or where mental symptoms are present, an operation should be performed, even though the dilatation of the veins be slight.

In the operative treatment of varicocele, which alone is curative, three methods may be considered. The subcutaneous ligation of the veins after the method of Keyes is still regarded by a few surgeons as the best way of effecting a cure in varicocele. For my own part, I do not believe in it and do not employ it in any case. The advantages claimed for it are that it is less dangerous, and that the patients quickly recover from the effects of the operation and are seldom confined to bed more than four or five days after the operation. In my opinion, the only real advantage is that no anæsthetic is required. This is of slight importance, because in cases where a general anæsthetic is contraindicated the open method can easily be applied by the use of the infiltration anæsthesia after the manner of Schleich.

Against these alleged advantages many arguments may be advanced in condemnation of it. In the first place, it is uncertain. Many cases are not benefited at all, or, if apparent recovery takes place, the cure is not permanent. Again, we might easily include in the ligature either the artery or the vas or both, or puncture a vein, any of which would cause some trouble. Furthermore, neither the cord nor the scrotum is shortened in this operation, conditions which are necessary for the permanent cure of all varicoceles of any considerable size.

Shortening the scrotum by removing a portion of it without ligature of the veins has been recommended and employed with success by a few surgeons. I do not consider amputation of the scrotum alone a rational method of treating varicocele.

The operation that I shall show you to-day, in treating the case before you, is the so-called open method of ligature of the veins supplemented by amputation of the redundant portion of the scrotum. This operation has the following points of superiority over the subcutaneous ligation of the veins: First, it is safe; the veins can be isolated and tied without any risk of including the vas or the spermatic artery and nerves, thus insuring, as far as the operation is concerned, the integrity of the testicle; the risk of infection is not much greater than in the Keyes operation. Second, this operation gives a permanent cure, even in the most advanced stages of the disease: relapses never occur if the veins are properly ligated and the scrotum is shortened.

The chief objection raised against this operation, that it necessitates confinement of the patient to bed for three or four weeks, does not hold good when the operation wound is kept clean. In cases where the scrotum is not excessive and therefore amputation is not required, I allow my patients to leave the bed in from three to five days. Where the scrotum has been amputated, a somewhat longer period of confinement to bed is insisted upon, usually from a week to ten days.

As to the question of atrophy of the testicle as a direct result of this operation for varicocele, I believe that it seldom occurs. The cause of atrophy when it is due to the operation is interference with the arterial blood-supply of the testicle by ligature of one or all of the arteries that pass along the cord to the testicle, or by division of the nerves of the cord. Division or ligation of the vas deferens does not cause atrophy. This has been demonstrated by experiments on animals, where the division was made for the purpose of studying its effects on the prostate.

The first step in the operation consists in making an incision about three inches in length through the upper part of the scrotum immediately over the varicocele. This exposes the fascia which incloses the cord and veins. The vas deferens, with its artery, and the spermatic artery and nerves are separated from the veins for a distance equal to the length of the incision. Around these is passed a strip of gauze, which is given in charge of an assistant, who holds them well out of the way. A catgut ligature is then passed around the veins, with the exception of one or two small ones which are allowed to remain, and tied close to the epididymis. A second liga-

ture is placed as high up as the upper angle of the wound will permit, and the veins are excised between these ligatures. The cut ends of the veins are now approximated and the ends of the two ligatures tied. A few fine catgut sutures are passed through the cut ends of the veins to insure approximation until healing is complete. All bleeding points are carefully sought for and ligatures applied. The wound is then closed by two layers of buried suture, one for the subcutaneous connective tissue and one for the skin.

In case the length of the scrotum is not deemed sufficient to warrant an amputation, shortening may be accomplished by uniting the upper and lower angles of the wound and suturing transversely instead of longitudinally. In this case the great length of the scrotum and considerable dilatation of the veins in it are sufficient indications for amputation. In order to avoid hemorrhage, the scrotum is drawn through a King's scrotum clamp. All the scrotum below the clamp is cut away with the scissors and the sutures are introduced and tied before removing the clamp. The patient does not lose any blood when this method is employed. An ordinary dry antiseptic dressing is applied, care being taken to keep the scrotum well elevated for the first two days after the operation.

## FIBROMA OF THE MALE URETHRA.

Case II.—The second patient whom I show you gives the following history: A. G., age twenty-four. Family history good; has had no illness except the diseases common to childhood. He denies having had any venereal disease. His present trouble began about one year ago, when he first noticed a small hard nodule about the middle of the penis. At this time the growth was not larger than a pea; it was hard and somewhat tender on pressure. About six months ago, after the tumor had grown considerably larger, he began to experience difficulty in urinating. This has gradually increased, until at present it requires considerable effort to empty the bladder.

Examination of the patient shows a well-nourished, strong young man, with all of the thoracic and abdominal viscera apparently in a normal condition. In the penis, about midway between the scrotum-penile junction and the end of the organ, there is a tumor about the size of a small olive, which is hard, slightly sensitive on pressure, and appears freely movable. This tumor is situated quite deep in the substance of the penis and is apparently attached to the urethra. On

introducing the sound we find the lumen of the urethra nearly obstructed by the tumor, which will not allow the introduction of a No. 2 English bougie.

The nature of the tumor cannot be definitely determined by the physical examination, but, from the history and from the points noted in the examination, it is probably a solid tumor of the urethra. That it is not an inflammatory swelling is evidenced by the manner of its development and by the absence of any history of a preceding inflammation of the urethra. There has not at any time been any discharge of blood from the penis, nor any considerable pain associated with the act of urination. This would argue against the tumor originating in the mucous membrane of the urethra.

The treatment of this condition consists in the removal of the tumor. An incision is made along the median line of the under surface of the penis through the corpus spongiosum down to the urethra, exposing the tumor, which is found to grow from the inferior wall of the urethra, and which by its growth upward towards the dorsum of the penis has nearly occluded the lumen of the urethra. Circumferentially the tumor involves about three-fourths of the urethra, which will necessitate the excision of about three centimetres of the urethra if the tumor is removed. The growth is carefully separated from the surrounding connective tissue, and the urethra close to the tumor, above and below, cut through. Two fixation sutures, one through the superior and the other through the inferior wall of the urethra, are used to approximate the cut ends. A soft rubber catheter is now passed into the bladder and the union of the cut ends of the urethra further strengthened by a row of interrupted sutures of fine silk. These sutures pass through all of the urethral wall excepting the mucous membrane. The incision in the corpus spongiosum is closed by a buried catgut suture.

The tumor removed is found to be oval in shape; its greatest length, that corresponding to the portion of the urethra excised, is a trifle under two centimetres. Its transverse diameter is a trifle less. On making a longitudinal incision through the tumor, we find that it grows from the submucous connective tissue, while the mucous membrane remains intact. The cut surface is smooth, has a grayish-yellow color, and is firm to the touch.[1]

[1] Microscopic examination of the tumor showed it to be a pure fibroma.

## RECTAL FISTULA.

Case III.—Our next patient gives the following history: C. F., male, age thirty-six. Family history good. Personal history of no particular importance. He has never had any other illness than that of which he now complains. His present trouble began about two years ago with severe pain in the lower part of the rectum. This was accompanied by a feeling of fulness in the lower bowel and was particularly severe during defecation. This pain had lasted for about one week, when a swelling appeared about the size of an English walnut on the left buttock near the anus. This was exquisitely sensitive to the touch and caused severe pain until rupture of the skin occurred, when immediate relief followed a free discharge of pus. Since that time there has been a constant discharge of thin pus, at times mixed with liquid fæces, from the opening, which still persists at the place where the abscess ruptured. Flatus occasionally escapes through this opening.

On examination, we see a small granulating surface about one inch behind and to the left of the anus. In the centre of this surface may be seen a small opening, from which on pressure a drop of pus escapes. On passing a probe into this opening, we find that it passes upward and to the right towards the rectum. I now introduce my finger into the rectum and search for the end of the probe. I find that it enters the rectum about three-fourths of an inch above the anal verge. Connecting the point at which it entered with the place of entrance into the rectum can be felt a hard fibrous band which corresponds to the canal traversed by the probe. We have, therefore, before us what is known as a complete rectal fistula.

In the consideration of fistulæ I wish first to direct your attention to the varieties that are most frequently met with. The most usual form is the so-called complete rectal fistula,—that is to say, there is a communication by means of a fistulous tract between the rectum and the external skin.

Of the complete fistulæ we find that there are three kinds, the division being based upon the location of the internal orifice of the fistula. The first and simplest form is that in which the fistula is subcutaneous, the internal opening being superficial to the external sphincter. This is the true fistula in ano, and to this form alone should this term be applied. In the next class the internal opening

is situated between the two sphincters. This is the most common of all fistulæ. The case before you belongs to this class. In these the position of the internal orifice is quite constant, usually being within one inch of the anal verge, at a point where the levator ani and fibres of the external longitudinal muscular coat of the rectum pass between the two sphincters. These fibres serve to direct the course of the pus as it forces its way into the rectum. Another reason why the opening into the bowel is so frequently found at this point is that this part of the rectum is subjected to the greatest amount of trauma in the passage of fæces, and is, therefore, the point at which perforation usually occurs.

The third form of complete fistula is above the sphincters. In this form the abscess is usually above the levator ani, and its point of communication with the rectum may be at any distance above the anus.

By an incomplete fistula we mean one that has but a single orifice, either mucous or cutaneous. Of these fistulæ there are two kinds,—the internal and the external. In the internal incomplete there is an opening into the rectum alone, the perirectal abscess having ruptured into the rectum without perforating the skin. Its origin is the same as that of a complete fistula, of which it is frequently only the first stage. If left without treatment it will in the majority of cases sooner or later perforate the skin and form a complete fistula.

The external incomplete fistulæ are those which do not communicate with the rectum, but which have perforated the skin at one or more points in the neighborhood of the anus. In some cases an opening into the rectum existed early in the disease but closed spontaneously. In others the abscess was located at some distance from the rectum and at no time communicated with it.

In passing a probe into a fistula we often find that it is not straight and that it varies in size in different parts of its course. These variations in size together with the frequent changes in its course may make the passage of a probe extremely difficult. In many cases there are diverticula given off from the main fistulous tract, or the internal orifice may be connected with the external skin by a number of fistulæ, which may open at any place around the anus or over the buttocks. In some cases the internal opening may be found on one side of the rectum while the external is on the opposite side, the fistula passing around a half or more of the circumference of the

rectum, usually just beneath the skin. These are the so-called horse-shoe fistulæ.

The structure of a fistula varies with the duration of the disease. In recent cases the tube is lined with fresh granulation tissue which rests upon practically normal structures. As time goes on the lower layers of granulation tissue change into dense connective tissue, which in very old cases is found in large amount and constitutes the indurated band that connects the internal and external orifices. At any time this tube may become obstructed, either from an exuberant growth of granulation tissue or from fæcal particles becoming lodged in it. The result is an acute inflammation with accumulation of pus above the obstruction. In such a case the old opening may close, either temporarily or permanently, when a new one or several new ones may be formed by the pus passing around the obstruction. The chief reason why fistulæ so seldom heal spontaneously is because of the presence of this dense connective tissue, which forms the outer layer of the wall of the perirectal abscess and fistulous canal and prevents their collapse after the pus has been evacuated.

For the examination of a fistula we place the patient on the side with the thighs well flexed on the abdomen. A small flexible probe is introduced into the external opening and carefully guided along the fistulous tract. The finger is then cautiously pushed into the rectum and the point of the probe sought for. In many cases the mucous membrane is separated from the muscular wall of the gut by the abscess for some distance above the internal opening, so that the probe may pass over it and only after a prolonged search made to enter the rectum. If the probe pass into the superior pelvi-rectal space and away from the rectum, so that the whole thickness of the gut is between the finger and the probe, we probably have to deal with an incomplete rectal fistula or a fistula originating from some other pelvic organ. If the internal opening cannot be found, we introduce a rectal speculum and then inject a small quantity of peroxide of hydrogen into the external opening of the fistula. If an internal orifice exists, its presence will be demonstrated by the peroxide foaming out through the speculum.

The recognition of an internal incomplete fistula is more difficult. Here we rely upon the history of pain and discomfort referred to the rectum, with frequent acute exacerbations followed by a more profuse discharge of pus. On examination, we find a point of induration on

the inner wall of the rectum which on pressure yields a greater or less quantity of pus. Around this a boggy area can be detected which is somewhat sensitive on pressure. Through the speculum we may be able to see the internal orifice of the fistula.

The symptoms occasioned by fistulæ vary greatly in individual cases. In the simpler form the patient complains of discomfort from the discharge of pus irritating the surrounding skin and from the occasional passage of fæcal matter through the fistula. In the more pronounced cases the oft-recurring attacks of acute inflammation from the obstruction of the fistula cause a great deal of suffering. The mental condition of the patient is frequently affected from the continued discomfort and many fruitless attempts at securing relief. He becomes despondent and suffers from loss of appetite and sleep, which, if continued for a considerable period of time, as is usually the case, seriously affects the general health.

In the treatment of rectal fistulæ palliative measures are of little value. In certain cases, where the patient refuses an operation or where his general condition contraindicates operative procedure, they may be employed. They consist in, first of all, regulation of the bowels and attention to the general health. In addition the fistulous tract is to be cleansed by frequent injections of antiseptic fluids. We may go farther, and attempt to promote healing by stimulating the growth of healthy granulations by the injection of astringent and cauterizing chemicals, such as chloride of zinc, carbolic acid, iodine, etc. Allingham favors this treatment in a certain class of fistulæ, and in addition recommends the introduction of a drainage-tube to promote free drainage and to stimulate the growth of healthy granulation tissue. This method is applicable only to those cases where the fistula is recent and where the tract is comparatively short and straight. Multiple fistulæ, or fistulæ with numerous diverticula leading from the main tube, and those which have existed for a long time, do not yield to this treatment.

The operative treatment that is commonly employed for the cure of fistula consists in dividing the tissues between the fistulous tract and the rectum,—that is, converting the rectum and the fistula into one canal. This operation has been practised by most surgeons during the last century, and on the whole is the best method of dealing with fistula. The steps of the operation are as follows: A probe-pointed grooved director is introduced into the fistula and the end

brought out through the anus. The tissues between the director and the rectum are divided, the incision being made either with a bistoury or with scissors. In case the internal orifice is so far above the anus that the director cannot be brought into the rectum, the incision is made on the director as far as it will reach and then completed by following the fistulous tract. Where there are several external openings with but one communicating with the rectum, each canal is laid open up to the internal orifice, making, if possible, but one cut through the sphincter. If more than one incision through the sphincter is required, it is better to cut but one (or two at most) at a time, to avoid the risk of incontinence. After the fistula has been laid open and the hemorrhage checked, the wound is packed with gauze and allowed to heal by granulation. The time required for healing varies from three to eight weeks, depending upon the depth of the incision.

The operation of excision of the fistula, which was brought into prominence by Dr. Lange, has many points of advantage. After passing a probe-pointed grooved director through the fistula and bringing the end out through the anus, I divide the tissues between the director and the rectum with the scissors. The fistula is then removed by a clean dissection, all of the indurated tissue being excised, from the external orifice to the highest point of the incision. The wound is now thoroughly cleansed, and the margins are approximated by silkworm-gut sutures, which are introduced in such a manner that the wound is closed throughout its entire length and depth. A piece of rubber drainage-tubing about three-fourths of an inch in diameter and five inches in length, wrapped in iodoform gauze, is introduced into the rectum. An ordinary antiseptic dressing and a T-bandage are applied.

The advantage of this operation is that in most cases we can secure primary union of the wound and thus shorten the time required in healing; there is also far less danger of incontinence resulting from it. Where there are several fistulæ, they can all be treated in the same way at one sitting if care be taken accurately to approximate the cut edges of the muscle.

Incomplete external or internal fistulæ we convert into complete fistulæ, excise the fistulous tract, and suture.

In some cases of short, straight, external incomplete fistulæ, or superficial complete fistulæ, curetting the tract thoroughly and packing with iodoform gauze may be sufficient to effect a cure.

# SHOT WOUNDS IN THE HEART REGION; SURGICAL TREATMENT OF TABETIC JOINTS; INGUINAL COLOSTOMY FOR SEVERE ULCERATIVE STENOSIS OF THE RECTUM.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF BERLIN [1]

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GENTLEMEN,—Our first patient this morning is a young woman of twenty-eight, who on Saturday afternoon last, six days ago, attempted suicide by shooting herself in the region of the heart. That such shot wounds are not always fatal those of you who have followed the clinics even during this semester know very well, and, if we may judge from the present tranquil, easy condition of this patient, the case is not at all likely to terminate fatally.

We get a number of these attempts at suicide here, and they nearly all conform to one of two types, head wounds and wounds in the region of the heart. They are not nearly so fatal as might be expected, considerably less than one-half the cases terminating in death. Wounds of the head are somewhat more frequent and also rather more fatal than those in the heart region, but recovery is by no means rare. Usually the revolver is pointed at the temple, and, as the skull is especially thin here, a fatal penetrating wound is often inflicted. It is surprising, however, how often even a bullet that enters the brain fails to cause death. Where it wounds only the frontal lobes, as may happen where the revolver is directed perpendicularly against the skull, there may even be no symptoms of injury of the nervous system, the silent convolutions giving no sign of their mutilation. It is wonderful, too, how tolerant important centres may be of bullet

[1] Reported by James J. Walsh, Ph.D., M.D.

wounds. Even the basal ganglia may be injured without immediately fatal results, almost without symptoms, and the patient die later from septic infection, from inflammatory exudate causing pressure upon vital nerve centres, or from pneumonia due to trophic influences caused by injury or interference with the function of the vagi.

That suicides by shooting do not oftener accomplish their purpose seems to be due to two reasons,—first, defective fire-arms or ammunition, and second, badly directed or at the last moment disturbed weapons. It would almost seem that manufacturers of revolvers and cartridges, with the philanthropic purpose in view of thwarting would-be suicides, made the ordinary classes of cartridges weak and the weapons inaccurate. Certain it is that they accomplish this very charitable object, whether intentionally or otherwise.

In firing into the skull the aim is often too much towards the front, and structures in the orbit are wounded or the bullet glances and finds its way out through the eyebrow, where the wound of exit may at first remain unnoticed or be taken for a wound from the fall after the shot.

Shots in the heart region are usually directed too far to the left to be fatal. If you take a revolver in your right hand and point it at your heart, you will find that it requires an effort of extreme wrist flexion, and a very inconvenient position must be assumed, in order to aim so as to perforate the heart directly. Most people use their right hands, and as in new weapons the trigger often pulls a little hard, the aim is disturbed in this constrained position. Then, at the last moment there is an almost involuntary movement of avoidance because of the pain anticipated. The trunk is revolved upon its axis towards the right and forward, the left side being withdrawn so that the bullet goes too far to the left. This change of position easily causes the bullet to glance on a rib and so fail entirely to penetrate the thorax. Most people, too, do not understand enough of their own anatomy to make successful suicides. While it is usually heart trouble that leads them to suicide, this is not of such a definite physical nature that its symptoms give them any clue to the position of the heart. These persons may have had palpitation of the heart, which perhaps was due to distention of the stomach, so that their notion of the position of the heart is vague and places it somewhere about the level of the stomach, certainly much lower than it really is. This often gives rise to complications in the shape of perforations of the

abdomen, which lead to peritonitis and death. Even when the bullet originally enters the thorax, it may afterwards perforate the arch of the diaphragm and injure abdominal organs.

Two things are to be remembered, then, as regards wounds in the heart region,—first, that the prognosis is not necessarily hopeless, that, in fact, more than one-half the patients recover; and, second, that it is not the heart nor the thoracic organs alone which may have been injured, but that in fatal cases exitus lethalis may be due to abdominal complications. This second part is important, for the symptoms of collapse due to loss of blood may be looked upon as fatal hemorrhage from the heart itself, when it is really due to the injury of an abdominal blood-vessel, for which surgical interference may be indicated.

In our case of to-day the wound of entrance is small, scarcely more than four millimetres (one-sixth of an inch) in diameter, and is, as you see, about midway between the ensiform cartilage and the nipple,—a little too low, perhaps, to wound the heart itself, though the right ventricle might, in case of slight dilatation, have been in the way. Penetrating wounds of the right ventricle are not so fatal as those of the left ventricle, the blood-pressure within it being less than one-third as high, though even wounds of the left ventricle are not invariably fatal, the thick muscular wall of the left ventricle furnishing a good hold for blood-clots which occlude the opening.

Nature has some wonderful resources at her command for the immediate closure of such openings. I have seen a case of shot wound almost in the spot where this one is, in which death took place more than a week after the infliction of the wound, with the symptoms of peritonitis. The autopsy showed that the right ventricle had been penetrated, the shot had then found its way through the diaphragm, opening the peritoneal cavity and wounding the liver, and had then passed *through* the aorta. Where structures are elastic, shot wounds push aside the tissues and pass between rather than through them. In such openings it is easy for nature to form a clot that will effectually plug the opening made, and it was this faculty which had averted the immediate death of the patient in the case mentioned, though there were four penetrating wounds of important parts of the vascular system, any one of which it would seem should be enough to cause an immediately fatal result.

The classical case of the stag in which after death there was found

distinct and unmistakable evidence, from the scars present, that the heart had been penetrated by a bullet, used to be referred to as a marvellous instance of nature's conservative power. Experiments upon animals during the last few years and more careful observation on the part of hunters show that the healing of heart wounds is by no means the extremely rare event it used to be thought. In the statistics of some five hundred cases of heart wounds in human beings some years ago, I remember noting that about twenty per cent. of them (and in more than one-half the cases the diagnosis had been confirmed by autopsy after death from other causes) had ended in recovery. Many of these were chance finds at autopsies, and the history of the case often showed that there had been no very serious symptoms at the time of the infliction of the wound, notwithstanding its intensely serious nature. Such finds are by no means so rare in the anatomical room, in these last few years, as is usually thought to be the case even by physicians.

In our case the wound that you see here, midway between the sternum and the nipple, was inflicted by an eight-millimetre (about thirty-two calibre) bullet. That the weapon was held close to the skin and was fired at the naked breast you can see by the discoloration produced by the remains of the explosive in the immediate neighborhood of the wound. The symptoms do not seem to have been at any time very serious. She did not lose consciousness nor did she faint after the infliction of the wound. She was found lying in pain on the floor of her father's room, into which she had gone for the revolver. She was brought to the Charité a couple of hours after the shooting, and, though breathing with some difficulty, was perfectly conscious at the time and has remained so ever since.

Her case was not, however, without its serious symptoms. There was, from the first examination here in the hospital, tympanites in the region of the heart, replacing the normal heart dulness. The normal heart tones could be heard, but there were murmurs added, murmurs occurring irregularly all through the cycle of heart sounds, and some of them very close under the listening ear. There were evidently air and blood in the pericardium, and these have not all been absorbed yet. I do not wish to disturb her by demonstrating the physical symptoms before you, but at the last examination it was noted that, while less than before, these symptoms of a pneumohæmopericardium were still present.

The bullet had evidently penetrated the pericardium, and the presence of blood in the pericardial sac might be considered to indicate that the heart itself had been wounded, though only superficially.

What are we to do in such cases? is the important question for the surgeon. Absolutely nothing except close the wound by an antiseptic dressing. It is worse than useless to attempt to render the bullet track aseptic; that has already been done by the heat of the projectile; and manipulation of the edges of the wound and of the sides of the canal would only increase the danger of infection, and perhaps disturb nature's well-arranged mechanism for the stoppage of the bleeding. To attempt to remove bits of clothing that may have been carried in with the bullet, or portions of a rib that have been broken off and carried some distance into the depths of the wound, is worse than illusory, since it can do no possible good and may easily do a great deal of harm.

The simple closure of the external wound, though it might not seem so, is often of good service in controlling the hemorrhage. It sometimes seems to give nature a needed point of support for her hæmostatic processes. I remember a case some years ago in which a man inflicted a stab wound some ten centimetres (two and one-half inches) deep in his fifth intercostal space on the left within the mammillary line. The withdrawing of the knife was followed by a spurt of blood, which he stopped by pressing his finger upon the wound. Whenever he removed his finger it spurted once more. He came to the hospital, still smoking a cigar, about half an hour after the infliction of the wound. Examination showed the presence of air in the pericardium and the irregular murmurs of pneumopericardium. Next day there was an increase in the area of heart dulness due evidently to blood in the pericardial sac. Closure of the wound was followed by healing by primary union, and the case ran an absolutely feverless course to complete recovery.

Where the heart is wounded, death usually ensues in fatal cases from the pressure of the blood which collects in the pericardial sac, disabling the already weakened heart. What to do in such cases when threatening symptoms supervene is a delicate question. We used to stand almost absolutely hopeless and powerless in the presence of these cases. The breathing would grow more and more difficult, as the needs of the organism for oxygen became more pronounced,

and the hampered heart failed to drive the blood through the lungs, and the pulse grew weaker and finally disappeared.

The first operative suggestion that was made for such cases was to tap the pericardium with a trocar, and, by letting out the blood collected in it, relieve the heart of the pressure that was so seriously hampering its action. This procedure has some good results to its credit and should not be given up entirely. Then came the incision of the pericardium, usually after the resection of one or more ribs; this, too, gave some very satisfactory results. But the real advance in the surgery of wounds of the heart has come in the last year or two.

It has been known for nearly ten years now that animals' hearts could after the infliction of wounds be sewed up and the life of the animal saved. The same, it was hoped, would prove true in the case of human beings. A Roman surgeon had the first opportunity to test the truth of the analogy. He inserted some stitches in a knife wound of the heart, and aborted the threatening fatal collapse that seemed inevitable. His patient died on the eighth day from pneumonia, but the autopsy showed that the heart was in progress of healing perfectly, and thoroughly justified the surgical interference in the case.

Then Rehn, of Frankfort, had a case in which serious symptoms developed on the seventh day after a stab wound in the heart region. He opened up the thorax and the pericardium, inserted five stitches in a wound of the heart from which blood was spurting at each systole, and his patient recovered.

For wounds in the heart region now, the position of the surgeon is one of armed expectancy. When the symptom complex described by Rose develops,—the faint or perhaps absent pulse, the distant heart sounds, the extreme dyspnœa, with deepening cyanosis, which indicate that the heart's action is hampered by the presence of blood in the pericardium,—the surgeon must open the thorax, clear out the blood in the pericardium, and take rational surgical measures to stop the bleeding, whatever may be its cause. That the number of lives saved by this operative interference, which a few years ago would have been thought eminently foolhardy, will be very large I have not the slightest doubt. This advance in the surgery of the heart and great blood-vessels will constitute one of the most interesting chapters in surgery in the next few years.

In our case we have never had to think of operative interference.

She came into the hospital in reasonably good condition and has constantly improved. The good condition of the patient, however, at the moment when the doctor is called, sometimes an hour or more after the infliction of the wound, must not lead you into expressing the opinion that immediate danger is over. Quite a characteristic course for wounds of the heart is first unconsciousness, or at least extreme faintness for a time, from shock, then a rally, during which the patient has scarcely any threatening symptoms, and then gradually increasing dyspnœa, faintness of pulse and weakness of heart's action, until the blood tamponade of the pericardium absolutely precludes the further possibility of dilatation after contraction—of diastole after systole.

There are some other dangers to be considered in our case besides those that come immediately from the heart itself. The tympanites replacing the heart dulness here in the region of the heart points to the fact that the lung was wounded as well as the heart. The peculiar murmurs heard are due to the same cause,—that is, to the presence of air and blood in the pericardium. At one time it was thought that the nature of these murmurs after injuries in the heart region would give a clue to the extent of the injury and to its character, but they may be due to such varied causes as valve injury, outpouring of blood, friction of the two planes of the pericardium, the presence of a foreign body, or even beginning pericarditis or the anæmia incident to the hemorrhage. Whether the murmur be "blowing, whistling, rubbing, sawing, metallic," or any of the other designations that have been found for it, depends largely on the amount of air and blood in the pericardium. No diagnostic importance can be attached to its character, and the surgeon must depend on the general condition of his patient, especially the pulse and the breathing, for the indications for operative interference.

Besides the air in the pericardium there was, the day after the wound, an area of dulness at the base of the left lung, evidently a hæmatoma due to bleeding from the wounded lung. That has not grown larger, however, and now there is very little danger of lung complications in the case. There is still some danger to be thought of from the peritoneum, for the shot was fired so low that it might have passed through the dome of the diaphragm, to find its way here to the lower border of the eighth rib behind, where I think we can detect its presence. Our patient has continually improved in every

way, however, so that now, on the fifth day, I think we need have little apprehension.

The main indications in these cases are rest and quiet heart's action. A large ice-bag over the left chest will often be of good service, and in strong, healthy patients, where the pulse is full, blood-letting may be practised. Morphine has a place also in the treatment and gives the patient the mental rest so necessary in these cases. Where the lungs are wounded, too, and the pain is severe, and the racking breathing develops which is so troublesome to the patient and leads to the increase of the pneumo-thorax, morphine is especially indicated.

Absolute rest is of the greatest moment, and must be continued until the pulse has been entirely and persistently normal for some time. I shall never forget the case of a young man in my service some years ago who had shot himself in the heart region. After four weeks he seemed to be so well that I allowed his mother to visit him. She proceeded to upbraid him for what he had done. The emotion that ensued was sufficient to undo all nature's reparative processes. There was a cardiac hemorrhage, and death followed in a few hours. Such an experience shows the imprudence of allowing relatives or others to talk to the patient with regard to the causes for his action. All violent emotion must be avoided in every possible way.

These patients are well worth a determined effort to save them, for, though they have attempted to take their own lives, the attempt is not liable to be repeated. The determined suicide is apt to take some effectual means at once, or he makes "assurance doubly sure" by taking poison and shooting himself. For many people an attempt at suicide seems the only suitable method of putting off the old love before taking on the new. It is often but the acme of the foolishness of a love story, and is not liable to be rehearsed again.

The case I have just shown you is one in which the surgeon's best course is to avoid interference except when threatening serious symptoms supervene. The lesson *non nocere* is an important one for the surgeon, so I shall show you another case in which abstinence from interference seems to me the best plan. An examination of our patient's pupils would show you that they are contracted. They do not react to light, though they do to accommodation. Her patellar reflex on the left side is absent, as you see. Her history would tell you the rest if you needed any further information as to her condi-

tion. She has tabes dorsalis. This large swelling at her knee on the right leg, which shows none of the ordinary signs of inflammation, which developed without pain and exists without tenderness, is a tabetic arthritis,—a serous effusion into the joint, due to trophic disturbance in the serous membranes, which occurs not infrequently in tabes.

Ordinarily one might think that with an effusion as large as this, indolent and persistent, it would be good surgery to evacuate it, but I have never thought that my patients got better any the sooner for it. I remember two tabetic patients who were sent to me last year because of large effusions. In one case, owing to the insistence of the patient himself, I tapped; the other I recommended to Hersing, the famous instrument-maker in Bavaria, for an apparatus that would enable him to be around and yet save the affected knee from further progress of the trophic changes, and from the slight injuries which lead up to these trophic changes in its state of lowered nutrition. A year later my second patient was in better condition than the first one.

In these cases care should be taken to avoid infecting the joint, for in its state of lowered vital resistance this would be extremely easy, and the patient should be provided with a suitable apparatus or with a good plaster-of-Paris bandage, which, while exerting continuous equable pressure, saves the joint from injury and keeps it perforce at rest.

Our next cases this morning are interesting examples of a condition whose etiology I think surgeons do not yet clearly understand. They represent two very different phases of the affection, the one in its post-active stage, the other just beginning, yet showing well the obstinate intractableness that often characterizes the affection. I refer to ulcers of the mucous membrane of the rectum, especially those which occur in women.

Very seldom do surgeons have the opportunity to observe the process from alpha to omega. They do not see the beginning, because the cases usually come into their hands when considerable loss of tissue, or at least a good deal of chronic inflammatory exudation, has taken place, which obscures the original condition. It is the custom to call most of them syphilitic; yet there seems to be no good reason why the syphilitic process should locate itself here so much more frequently than it does in any other part of the gastro-intestinal

tract. At most, its favorite locations should bear some relation to those of cancer, if the ordinary slight injuries, etc., are the immediate cause. Then, too, syphilis is more frequent in men, while the intractable rectal ulceration I refer to occurs almost exclusively in women.

That these ulcers are venereal in origin I have not the slightest doubt, and I am inclined to think them due to a primary gonorrhœal affection of the rectal mucous membrane on which a secondary infection with pus cocci had been inoculated from the intestinal contents. In women the relation of the anus to the posterior commissure of the vagina is such that it can be easily understood how gonorrhœal infection might take place by the flowing of infective secretion over the perineum on to the anus. This would account for its frequency in women.

It is usually in women of doubtful character that it is seen, though at times it will be found in members of the best families. After all, respectability does not make the tissues a less favorable culture medium for the gonococcus, and accident and conjugal infidelity play a *rôle* in the mediate etiology.

That it is not, as a rule, due to pæderastic practices I am firmly convinced. If it were, we should find it oftener in the passive pæderasts of the other sex. Besides, it is not usually accompanied by the dilated anus, with pouting, hyperæmic mucous membrane, of the pæderast. The venereal infection is not direct, but an indirect one from the vaginal secretions, as we have said.

Our first patient, aged forty-two, unmarried, is the mother of an illegitimate child. Ten years ago she had syphilis, for which no remedies were taken for three months. As the ulcers in the genital regions became very severe, she was under treatment off and on for a year; but she was not free from ulcers, especially of her labia minora, for some years. About three years ago she had a severe fluor albus. This was treated, but there were various relapses of it, and it gave her a great deal of trouble. Shortly after this she had pain and difficulty at stool, and noticed that her fæces were streaked with blood and pus. She seems to be one of those peculiarly insensible people, with undeveloped imaginations, who think that life means suffering anyhow, and we must grin and bear it, and so she did not consult a doctor. She made her stools soft by an occasional purgative or a cold water injection. This made the condition bearable for a few days each time.

Over three years ago she developed incontinence of fæces, and has had to wear a pad ever since. Some three months ago she developed also incontinence of urine. When you see the condition that is present you will not wonder. The only cause for wonder is that the sphincters did not sooner refuse to perform their functions. Everything here in the perineal region is affected. The ulcerative stenosis led to fistulous tracts all around the anus. Some of these opened into the vagina. The ulcerative process sank deeper and deeper into the tissues along the fistulous tracts. The perineal body was eaten away entirely. The labia majora were attacked, undermined, and loosened in flaps as you see here. The labia minora melted away before the ulcerative process. Finally the urethra was attacked and the anterior vaginal wall. The partition between the bladder and the vagina has begun to go, hence the incontinence of urine. And all this has been allowed to go on almost without a complaint!

The ulcerative process is not now very active. Most of these surfaces are cicatricial tissue. There are absolutely no signs of syphilis at present, nor have there for years been any except this that might be considered syphilitic. There is now here a continuous discharge of foul matter from the rectum. Rectal examination shows the presence of granulation tissue up to the stricture. The stricture itself is very tender, but beyond it the infiltration does not seem pronounced. From the vagina, and in women it is from here always that the presence of periproctal infiltration can be best judged, the rectum below the stricture feels like a thick, hard rope.

When it came to the question what to do here, the first thing I decided upon was that the passage of fæces over this ulcerated tract, irritating and still further infecting it, must be stopped. Of late there have been afternoon rises of temperature, indicating septic absorption, and the process would seem to be invading parts of the rectum from which infection of the peritoneum might take place. With this idea, a week ago I made an artificial anus by inguinal colostomy, and that is the reason why the ulcerative process is now so much better. I shall let it heal still further and then decide what can be done by a series of plastic operations, when you shall see the case again.

Our second case is a girl of but nineteen, the daughter of a member of a troupe of "specialty theatrical artists." About two years ago she probably had syphilis, at least there was an eruption that seems

to have gone back under antisyphilitic treatment,—*i.e.*, under a rubbing cure, as she says. Since then there have been no further signs of syphilis. Shortly after her syphilitic symptoms she had a severe leucorrhœa, with a plentiful whitish-yellow discharge. A little later she noticed difficulty and pain at stool and blood and pus in the evacuations.

She allowed this condition to go on for about a year, and then, some four months ago, came to us. We found an ulcerative stricture of the rectum, uncomplicated by fistulous openings, and the upper edge of which I thought I reached without much difficulty. The process was an extensive one, and the walls of the rectum were deeply infiltrated. We treated it, as we usually treat these cases, by cauterization with pure chloride of zinc. This is applied on a piece of cotton, allowed to act for half a minute, and then washed off. For the pain caused by the procedure I give morphine. Every ulcerated patch must be touched, however, or the granulating surfaces after cauterization will be reinfected. The bowels are thoroughly emptied before the cauterization and then allowed to remain closed for three or four days. When evacuations occur, cleanliness is secured by irrigation with sterile water or normal salt solution. After three or four weeks, if there is still some pus in the stools, a second cauterization is done. Usually, with care to touch all the affected parts at the first cauterization, I have not found the repetition of it necessary.

In this case it failed to give complete relief even when repeated. After three months in the hospital a more thorough cauterization was done; then for a time she seemed better. For the holidays she left the hospital and went home; after two weeks, however, she returned with the old trouble. Now, besides, we find that there are afternoon rises of temperature. Though she is thin and delicate and has lost in weight of late, there are absolutely no signs of tuberculosis of the lungs. The fever is evidently due to the absorption of septic products from the rectal ulcers, and something must be done to stop it.

In my opinion, we have here, as in the other case, not a syphilitic process, but the results of secondary infection with bacteria from the intestine after a primary gonorrhœal infection. Antisyphilitic treatment has done neither of them any good. To prevent further irritation by the passage of fæces and give the part the physiological rest so conducive to healing, I shall here, as in the other case, make an artificial anus. She is an extremely young subject for so serious a

procedure; but the obstinacy of the affection to treatment, its tendency to spread and become destructive, as in our first case, and, above all, the septic absorption, with febrile symptoms, that it is causing at present, thoroughly justify serious surgical measures.

I shall do an inguinal colostomy, as in our first case, and, after the patient has recovered from the operation, we shall carefully cauterize the rectal ulcers, and hope then to have them heal without further delay.

When the descending colon was exposed for the purpose of making the artificial anus, it was found that points of infiltration existed so far up that the opening in the gut had to be made a little higher than usual to be sure of being in healthy tissue. This spread of the affection upward showed clearly the necessity for the surgical means employed.

# THE LATERAL ROUTE FOR THE REMOVAL OF CERVICAL GROWTHS.

BY EDMUND W. HOLMES, A.B., M.D.,

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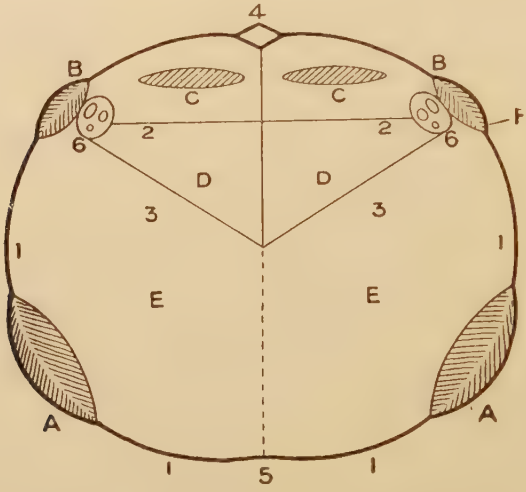
The average practitioner has a wholesome dread of the lateral region of the neck, by reason of an ill-defined impression of the proximity of the greater vessels and nerves and an absolute lack of knowledge of the distribution of the cervical fascias.

The median incision and its variations are usually preferred for the removal of growths approaching the median line, with a resultant injury to the pretracheal muscles and the vessels and nerves supplying them, with the disadvantage of a comparatively narrow area for operative procedures, with the uncertainty of working towards the great vessels, with the greater difficulty in the control of hemorrhage, and with far less facility for drainage. Tumors rarely begin in the median line, but their natural tendency is inward, because the motility of the larynx and œsophagus necessary to function renders that the direction of least resistance, while the firm cervical fascia acts as counter-pressure. The lines of deep cervical lymph-glands are inviting sources of abscess and of sarcomatous growths. Caries of the vertebra may cause pus collecting in the vicinage. Lymphangiomata, angiomata, blood cysts, carbuncle, and bronchocele in this region not infrequently demand the services of the surgeon.

I have often deplored the lack of attention given to the fascias of the neck, and have frequently expressed my admiration of the wonderful provision of nature by means of which the prevertebral fascia (3), holding back the prevertebral muscles against the spine, affords a sliding plane for the free expansion and movements of the œsophagus and larynx, and equally a smooth tract for operative procedures; while the sterno-mastoid muscle in its separate sheath (B) can be

lifted off from it and from the subjacent vessels (6), and can readily be retracted towards the median line, leaving abundance of space underneath. Further, laterally we have valuable bony reference points,—the apices of the transverse processes,—just within which lie the carotid artery, the internal jugular vein, and the vagus nerve, (6) in the vallecula between the muscular masses, while the intervertebral notches between the transverse processes transmit the cervical spinal nerves. Moreover, as the branches of the superficial cervical plexus are quickly distributed to the integument and the platysma, only a few of the descending branches are cut by the lateral incision,




THE DEEP CERVICAL FASCIA.—1, investing fascia of neck; 2, pretracheal layer; 3, prevertebral layer; 4, space of Burns; 4-5, median line of neck; 5, spinous processes of vertebræ; 6, carotid artery, internal jugular vein, pneumogastric nerve. A, trapezius muscle; B, sterno-cleido-mastoid muscle; C, depressor muscles of the larynx; D, trachea, œsophagus, thyroid gland and isthmus; E, prevertebral muscles and vertebræ; F, line of incision.

while in the usual dorsal decubitus free drainage is thoroughly accomplished.

We have operated for abscess, sarcoma, and bronchocele by this route, and are satisfied that for all deep-seated growths below the level of the upper margin of the thyroid cartilage it affords a more satisfactory means of approach than the median incision, giving a better command of the field of operation, with more room, greater facility in the arrest of hemorrhage, to say nothing of the ready avoidance of the great vessels of the neck, and far better drainage.

The following case of fibrous bronchocele well illustrates the

points presented. The patient, fifty-six years old, about eight years ago noticed a small swelling upon the right side of the neck at the outer edge of the lateral lobe of the thyroid gland, which very gradually increased to the size of a goose-egg, growing inward until it displaced the larynx about an inch to the left of the median line. During the past year he had some dyspnœa, and during the past month beginning dysphagia. Medical treatment proving of no avail, the increasing difficulty in breathing and swallowing drove him to an operation.

After the usual aseptic preparation and etherization, the head was rotated to the left and a four-inch incision made (F), parallel with and upon the posterior edge of the right sterno-mastoid muscle, avoiding the external jugular vein; the muscle was then lifted with a retractor, and the carotid artery and accompanying vein were found at its outer edge, having been forced outward by the growth; the pretracheal fascia (2) being cut, the tumor was seized and enucleated as far as possible with the handle of a scalpel; the arterial branches above and below from the superior and inferior thyroids being tied off, the remaining slight bleeding was chiefly venous. The depressor muscles of the larynx having been lifted upon a retractor, the mass was under complete control, and the isthmus of the thyroid was rapidly approached and separated from the surrounding structures. There were no adhesions to the œsophagus. The isthmus was then transfixed with chromicized catgut and tied, first in two parts and then encircled as a whole, and the pedicle cut through. After douching with hot water, the wound was closed with a rubber drainage-tube at the lower angle. It is evident that one lobe of the thyroid remained.

In another patient with a cervical sarcoma, a couple of years ago, after a similar primary incision the malignant growth was found to be universally adherent. The left common carotid artery was tied, the greater part of the left sterno-cleido-mastoid muscle was removed, the tumor being dissected from the submaxillary and parotid fascias; but, in spite of this extensive dissection, the patient made a rapid recovery and was capable of movements of the head and neck, which rather controverted the usual ideas of the function of the muscles destroyed; she died, however, six months afterwards from a recurrence.

In another case in which I operated some time since, the growth

is already returning. The peculiar history this patient gave me was that the swelling appeared suddenly one night upon the left side of the neck. It was diagnosed as an aneurism by a very capable surgeon; for myself, from the fact that the man had had a recent attack of catarrhal pneumonia, from the rapid onset (though I never rely much upon "history"), from watching the growth in the ward and seeing it increasing from day to day, with reddening of the skin and rise of bodily temperature, and from its indistinct fluctuation beneath the sterno-mastoid, I diagnosed it to be a tubercular abscess. The operation showed it to be a cystic sarcoma. This is not as bad an error as that of Liston, who mistook, it is said, an aneurism in this region for an abscess, plunged in his knife, and the patient promptly died from hemorrhage.

# THE VARIOUS FORMS OF TALIPES; CONGENITAL ABSENCE OF THE FOREARM AND LATERAL CURVATURE OF THE SPINE.

CLINICAL LECTURE DELIVERED AT THE VANDERBILT CLINIC, NEW YORK.

BY VIRGIL P. GIBNEY, M.D.,

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Gentlemen,—I shall first show you a young man upon whom I operated a week ago because of very exaggerated cavus, which made his foot nearly as badly deformed as that of a Chinese lady, and compelled him to walk almost like a chicken. You will remember that, while my assistant made pretty strong traction on the toes, I divided the tendo Achillis and other contracted parts. The operation itself is usually devoid of pain if you have all the conveniences. The main thing to avoid is swelling of the toes from too tight bandaging. Don't be afraid that you will make the tendon too long or too weak. The great objection to tenotomy on the part of the laity is due to the belief that the tendon is weakened by dividing it. It is for this reason that you will find it so difficult, no matter how slight the operation itself may be, to get consent. It is hard to tell why this impression prevails, except that in a few cases (spastic equinus) in which the operation has been done, the spasm has affected the anterior muscles, and the patients have walked worse than before. They are, however, only the exceptions which prove the rule. I see no reason why the tendo Achillis should not be divided freely, and the foot brought down a little beyond ninety degrees. Some surgeons after division of the tendo Achillis leave an interval between the divided ends of from one-half to one inch. In drawing your bandage over this space, be careful not to let it sink in between the ends of the tendon. You should place a pad on either side and then apply the bandage in a figure-of-eight so that it will not fill in this space. You desire to have this space filled in with blood, which becomes organized and unites the divided ends

of the tendon firmly. If you neglect this precaution, your patient will have a weak tendon. This man has a plaster-of-Paris dressing and a board incorporated in the plaster bandage over the sole of the foot. He will wear this dressing for a week longer, and then I shall apply an apparatus consisting of a foot-plate and an upright. If the toes show a tendency to draw up, I shall pass a few turns of webbing through the slots in the distal end of the foot plate, and counteract the upward tendency in that way.

The right foot of this same patient is supposed to be sound and normal, but when I ask him to make dorsal flexion he cannot do it to any extent. The anterior tibial muscle draws the metatarsal bones almost vertically instead of horizontally. The tendo Achillis is also shortened. After a time, when the other foot is better, we shall operate upon this tendon, and also divide the plantar fascia. The loss of power in the anterior tibial group is shown by the way he lifts the foot in walking.

Here is a little girl of four years who was born with club-foot of the right side. She has now what is known as relapsing club-foot. A year ago she came to the clinic and was operated upon by the chief-of-clinic. The foot was restored to a good position, but we lost sight of her for several months and now find her in this condition. The tendo Achillis has shortened again and the plantar fascia is tense. The foot can barely be brought up to a right angle.

Here is another case. When this boy was eleven months old he had a hemiplegia of the right side. He lost the use of his hand and had trouble with the vision of the right eye. In order to get the right heel to the floor he is obliged to bring the knee back into a position which we call "recurvatum." This condition is found in many cases of equinus, and even in some which are supposed to be cured. In this position photographs may be taken of the patient showing the foot flat on the floor, but if the photograph be taken in such a way as to show the knee in recurvatum the fallacy at once becomes apparent. The explanation is simple. You know that the gastrocnemius is attached to the femur and to the os calcis, and in order to get the heel down it is necessary to throw the knee back. The principle of subcutaneous division of the tendon is that the blood flows out of the divided ends and fills in the gap; the blood then becomes organized: at least that is a theory which has been quite generally accepted. At any rate, the tendon becomes renewed, and

microscopical examination a few months afterwards fails to show any difference between the new portion of tendon and the undivided part. This boy needs to have the tendo Achillis divided and the foot put up in plaster of Paris. There seems to be a hopeless paralysis of the anterior tibial group of muscles. After the healing of the tendo Achillis it will be found that these muscles can be moved with much more force; it is rather a weakness than a paralysis of the muscles. This is one argument in favor of dividing the tendo Achillis.

You notice that when this next little boy walks he strikes his heel first and then the foot comes down in a peculiar manner. He is five years of age, and has been walking in this way since last August. In July he had a fever, and after this the mother noticed the change in his gait. In all cases of defective nutrition or paralysis one can discover at a glance a difference in the "expression" of the two limbs. It does not attract the attention of the novice, yet, if there were such a marked difference in the two sides of a person's face as there is here between the two lower limbs, you would all notice it at once. In this case the tendo Achillis on the left side appears relaxed and poorly marked, and there is atrophy of the muscles on that side, making a difference of three-fourths of an inch in the circumference of the calf on the two sides. He has some power left in the gastrocnemius group on the left side. Our aim in this case must be to keep the foot extended and give the tendo Achillis an opportunity to shorten. One of the best results that I have obtained in a case of calcaneus in a young child followed retention of the foot in an extended position, by means of plaster of Paris, for a year and a half. Plaster of Paris was used because I had great difficulty in keeping on any apparatus which the child would not break. The shoe was made to go over a wedge-shaped piece of cork and was provided with a very long heel. At the end of a year the tendon was fairly strong, and at the end of a year and a half it was fully as strong as the other. The boy now wears an apparatus to prevent straining the tendon. This is a mere suggestion regarding the non-operative treatment of talipes calcaneus.

The operative treatment is begun by making a Y-shaped incision through the skin down to the tendon. The V-shaped portion of the "Y" is then lifted up and dissected back to the muscular portion The stem of the "Y" is separated from the tendon, and the latter is exposed for about two inches. With a retractor slipped under the

tendon it is raised out of its bed. It is shortened by oblique section and overlapping, and then several "quilt sutures," preferably of kangaroo tendon, are passed through the portion overlapping. You can then still further separate the stem of the "Y" and by forcibly extending the foot make a V-shaped wound, which should be sewed up with catgut, and it is a good plan to pass every other suture through the tendon. I can hardly lay too much stress upon the importance of getting a dense cicatrix here, because if this is not obtained the tendon will stretch and the calcaneus will recur. This is about the best operation on the tendon. A certain number of cases have been cured, and of the fifty per cent. of relapses many, I believe, were entirely the result of a failure to do a thorough operation.

As I am desirous of showing you the different varieties of talipes to-day, I will next direct your attention to this little child, who presents the condition known as talipes valgus. The arch of the foot is lost. There is a calcaneus on one side and valgus on the other. Here is a little child with what may be called another form of talipes; it is a case of rachitic flat-foot.

The next case I wish to present is one of congenital absence of the forearm. The patient is a young woman who has only a small stump projecting below the right elbow where the forearm should be. She is at present wearing this artificial hand. The apparatus is very light and gives her a good appearance. She comes to us to-day, however, not because of this peculiar congenital deformity, but because of a lateral curvature. It is only in the upper dorsal region, and the twisting is towards the left, as is evident when she bends far forward. When she is in this position, one can easily see that the ribs on the left side are more prominent than those on the right and that the spinous processes of the vertebræ look towards the right. Her congenital deformity has weakened the left side, and, as a result, she has developed this condition of the spine, which is known as a compensatory lateral curvature.

# THE NECESSITY FOR THE EARLY RECOGNITION AND PROMPT REMOVAL OF ALL SO-CALLED BENIGN TUMORS IN THE FEMALE BREAST.[1]

BY WILLIAM J. TAYLOR, M.D.,

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It would hardly seem necessary for any one at this day to write a paper insisting upon the need for the early and complete removal of all new growths from the female breast, no matter how small or insignificant they may appear, and I would not bring the matter before you to-night were it not for the fact that we constantly see the results of neglect on the part of the general practitioner of this wise and imperative rule. Frequently a woman will present herself with a lump in her breast which she had discovered some months or years previously and for which she had consulted her family physician. He has told her that it is a very small affair, that it amounts to nothing, that she need not worry about it, and that all that will be necessary for her to do is to use some simple ointment which will absorb it; that if it were malignant, or at all likely to become so, it would give her pain. Again, if this use of the ointment has not absorbed it in the course of a few weeks, he may put a little cocaine under the skin and cut the lump out without, however, any special surgical precautions. This is the history frequently given to the surgeon, and when the patient is seen and a careful examination made, the mischief has been done, the growth so far advanced and the tissues of the breast so deeply involved, that only a very extensive operation can give any hopes of eradicating the disease, while frequently all that can be expected is a prolongation of life. Small benign tumors may be met with in young women,—indeed, in women approaching middle life,—but it is at about thirty years of age that we find the greatest number, and with the exception of a

[1] Read at a meeting of the Philadelphia County Medical Society, May 24, 1899.

few cases of fibroma and the exceedingly rare lipoma, they are not to be expected. If it is by any chance an adenoma, which under the older teachings was considered a benign tumor, it will almost inevitably in the course of a few years become malignant; and, indeed, certain of the Germans consider that all adenomata are merely the first stages of carcinomata.[1] Mr. Warrington Howard (in the *Lancet*, 1894, vol. i. p. 1245) reports one case of an adenoma of the breast which had persisted with very little change for nine years, but that at the end of that time it became malignant, and Mr. Jonathan Hutchison, Jr. (Transactions of the Pathological Society, London, vol. xxxix. p. 319) gives other instances. The imperative need for early operation is well shown in a case under my own care some three years ago. This was a woman, unmarried, aged forty, in good health, who discovered in November a small lump in her left breast. She consulted me the end of December, when I found a small, perfectly smooth mass about an inch in diameter which gave no pain, was not tender, was not attached to the skin, and which was apparently a perfectly innocent growth. It was impossible for certain personal reasons to operate until the beginning of January. The growth was found to be about one inch in diameter, round, within a capsule which was so distinct that the growth itself could be peeled out without the slightest effort. It had no attachment to the rest of the breast, and in view of its recent growth, its apparently perfectly innocent character, and of her occupation, which was that of a music-teacher, in which she needed perfect freedom of the movements of the arm, it did not seem wise to remove the breast itself. A very careful microscopic examination of the whole growth was made by Dr. D. Braden Kyle, including the capsule in its entire circumference. The growth was an adenoma, which in its centre had undergone malignant changes, but this had not yet progressed

[1] S. W. Gross (Tumors of the Mammary Gland) classifies tumors of the female breast as follows: Fibroma, lipoma, and chondroma as absolutely benign; myxoma as semi malignant, as it shows a tendency to recurrence or reproduction after removal; sarcoma as excessively malignant; carcinoma, of course, malignant; adenoma he speaks of as eminently recurrent and that it starts from the lacteal glands, as does carcinoma; it is rarely met with.

Roger Williams (Diseases of the Breast, p. 128) found in a total of 9227 cases of neoplasms in females that 26 per cent. occurred in the breast, and in 2397 consecutive cases of primary mammary new growths in women 1863 were carcinoma, 92 were sarcoma, 2 were myxoma, 372 were fibro-adenoma, 3 were papilloma, 1 was fibroma, and 63 were cystoma.

sufficiently far to reach the capsule in any direction, nor was there any evidence at any point in the capsule of any malignant change. If any persistent efforts at dispersing the tumor by ointment had been advised, it would have caused such delay that at the time of operation the whole breast must have been sacrificed to save her life, whereas with the results of the microscopic examination it seems perfectly fair to presume that the disease is entirely removed, and she will never have any further recurrence of it. It is now three years and there is no sign whatever of any further trouble.

Another instance, also in a woman of forty and unmarried, who had a similar lump in her breast, consulted her old family physician, who made light of the matter and told her that it could be absorbed readily by ointment. This she tried faithfully for several months, without any benefit. When it did come to operation, some six months after the time at which the growth was discovered, it was found that a similar growth existed within a capsule almost as distinct as in the previous case, but that the delay had been such that malignant changes had occurred, and had pushed their way through the capsule, which it had invaded, and had affected the whole of the structure of the breast itself. It was necessary not only to remove the breast but also the pectoral muscles and axillary contents.

These are but two illustrations of a number of cases which have come under my observation within the past few years, and I cannot urge too strongly upon the general practitioners who first see these cases the necessity for early and complete removal of all new growths in the female breast at no matter what age they are first noticed.[1] If it is in a young girl, it is probably a fibroma, or really fibro-adenoma, and as a rule perfectly innocent. It is a very simple matter to take this out without destroying in any way the function of the breast, and it does away with the possibility of future danger. As the age increases up to the time of the functional activity of the breast, it is all the more necessary that very small growths be gotten rid of, and if approaching forty, or beyond, it is absolutely necessary to remove the growth at the earliest possible moment of its discovery,

[1] Roger Williams says that the age at which carcinoma of the breast developed in five hundred cases varied from twenty-four years as the earliest to eighty-four years as the latest, with forty-eight years as the mean. Of this number 0.6 of one per cent. developed under twenty-five, 4 per cent. developed between twenty-five and thirty, 6 per cent. developed between thirty and thirty-five.

while if there is the slightest suspicion in the mind of the surgeon of malignancy the whole breast must be taken, with the contents of the axilla and the pectoral muscles.

I have purposely not mentioned cases of cystic degeneration of the breast, which we see so frequently and which should be treated without exception by early and complete removal of the whole gland.

Pain as an early symptom of malignant disease of the breast is most uncertain, or rather, I should say, very unusual, for in my own personal experience, which is now very large, I have ceased to rely upon it as in any way bearing upon the diagnosis. It is only later in the disease, when the tumor has grown large or where the breast is extensively involved, that it becomes of any importance.

I would therefore offer the following conclusion:

First, all cases of tumor of the female breast must be immediately submitted to operation and a thorough removal of all of the new growths. If this be absolutely limited and encapsulated it may be sufficient to remove the growth alone, but it must be examined by a competent pathologist, and if there be the slightest suspicion of malignancy or involvement of the capsule, then the whole breast must be sacrificed.

Second, if the growth appears on section to be an adenoma and attached to the tissues of the breast, the whole gland must be removed, the axilla cleaned out, and at least the pectoral fascia cut away, if not both pectoral muscles.

Third, wherever it is practical, an immediate microscopic examination should be made at the time of the removal of the growth, and this can be very well done by using a freezing microtome and hardening the section with formaline. The whole process does not take more than fifteen minutes. If the report from the pathologist is a negative one, nothing further need be done at that time; but, on the other hand, if there be even a possibility of malignancy or of degenerative changes in the tumor removed, even if it does not show distinct malignancy, it is not safe to run the slightest risk,—the patient must be given the benefit of the doubt and the whole breast removed.

In this short paper I have purposely not taken up the general subject of malignant disease of the breast, for I think we now all agree upon the needs for early and radical operative procedures. By this I mean the sacrifice of both pectoral muscles, major and

minor, the careful dissection of the axilla and removal of all of the fatty tissues surrounding the great vessels and nerves and the lymph-channels with the microscopic glands, for even if no glands can be felt by the fingers, their infection may have occurred,—an infection which can only be demonstrated by a systematic microscopic examination of all the tissues removed.

Then, too, if there be even a suspicion of glandular involvement, an incision should be made above the clavicle and the whole of the fatty mass in the superior clavicular triangle dissected away.

Although this would seem a pretty radical measure to advocate, it is surprising how little it increases the danger of the operation itself, and if the operator and his assistants be clean, the convalescence is not prolonged, and I do not think the mortality in any way increased.

# FRACTURE AND DISLOCATION OF BONES OF FOREARM, WITH COMPLETE PARALYSIS OF THE HAND; VASCULO-PARENCHYMATOUS GOITRE.

CLINICAL LECTURE DELIVERED AT THE NORTHWESTERN UNIVERSITY MEDICAL SCHOOL.

BY FREDERICK C. SCHAEFER, M.D.,

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ASSISTED BY

DRS. A. A. BRISLEN AND DANIEL ROGERS.

Gentlemen,—Our patient, Mr. J. A., is thirty-six years of age. He is a strong, muscular man, five feet and ten inches tall, and weighs one hundred and eighty pounds. On September 29, 1897, he fell from the top of a tower one hundred and eighty feet high, on to an iron beam twenty feet below the crest of the tower, striking on the ulnar side of his right arm; he then swayed away ten feet from the iron beam and fell across a staging twenty feet lower. He held on to the staging with his left hand, his right hand being useless. His fellow-workmen came to his rescue; they lowered him to the ground, and sent for a physician, who discovered a dislocation of the right foot, also a fracture of the right ulna and a forward dislocation of the right radius on to the humerus. The dislocation at the ankle was reduced at once. The arm was greatly swollen and bruised. It was promptly treated by the attending physician, and twelve hours later the patient was sent to Chicago.

It is now fifteen weeks since the accident occurred. The condition of the bones is indicated by the X-ray picture. The hand is paralyzed completely, except that he can flex the middle and ring fingers about one-half inch. There is absolutely no sensation in the entire hand or wrist; sensation ceases at the lower end of the radius and ulna, and on the radial side of the arm two inches above the

FIG. 1.—X-ray of right arm, showing fracture of ulna and forward dislocation of radius.

styloid process of the radius. A week ago the hand was in contact with a hot stove, but the patient was unaware of it until he smelt the odor of burning flesh. He can supinate and pronate the arm to a slight extent, but has no other movement. He cannot feel the needle point anywhere about the wrist, hand, or fingers. He is not conscious of having a hand. The arm feels as if it were constricted by a bandage in its upper third; he has no thermal sense anywhere in the area mentioned. The fingers are rigid in semi-flexion; the skin is dry, harsh, and cold; the small muscles of the hand are atrophied; the thumb is in a straight line with the outer border of the radius in extension. There is a very large callus at the juncture of the upper with the middle third of the arm at the inner and posterior aspect. The arm bows at this point, with convexity looking inward and backward. Although sensation is perfect in the forearm, he is not able to move any of the flexor or extensor muscles.

The history of the case and the condition at the present time indicate serious injury to three large nerves, and possibly to five. The nerves involved are the median, the ulnar, and the musculo-spiral, also the radial and posterior interosseous, separately.

The manner of the accident in all cases of injury should be closely inquired into, as it frequently aids one in making a diagnosis. This man fell first twenty feet, striking with the full weight of his body against his right arm; the ulnar side of the arm, in all probability, impinged on the edge of the iron beam. The blow was an extremely powerful one, sufficiently severe to break the ulna, force the fragments against the radius, and throw the latter bone out of its socket at the elbow. To my mind the indications are plain that the fracture occurred first and the dislocation instantly followed. After the radius was forced out of the socket, it was probably lifted an inch or more away from the humerus, and must have stretched the musculo-spiral nerve which runs along its inner anterior aspect, and I believe the stretching of this nerve, with the possibility of subsequent pressure of the head of the radius upon it, is sufficient to account for the paralysis of the extensors of the forearm and hand. When the nerve was stretched some of its fibres must have been torn; it is also possible that the posterior interosseous nerve is implanted in the callus, but the fact remains that there was instantaneous loss of motion and sensation at the time of the accident. There must, then, have occurred injury to the musculo-spiral nerve before its division

at the elbow. The median and ulnar nerves, I believe, were damaged by the impingement of the ulna against the iron beam, or by the fragments of the fractured ulna, which were driven obliquely outward against the radius, contusing or lacerating the nerves. The contusion of a nerve would be sufficient to break the nerve-fibres without even lacerating the sheath. The ulnar nerve may have been directly damaged by forcible contact with the iron beam, as it lies nearer the inner side of the arm. Probably all of the nerves were immediately damaged in the manner described; but I feel confident also that subsequent injury has occurred to a part of them. Undoubtedly great damage was done to the soft parts of the arm; the fracture occurred at the fleshy part of the arm, the muscles and aponeuroses were lacerated, and in the regenerative process which followed a great deal of cicatricial tissue was produced. What effect bands of tissue may exert upon the nerves can only be conjectured before operation.

The patient is anæsthetized and prepared for operation. I will, therefore, proceed to cut down upon the median nerve in the middle portion of the forearm. The first incision is five inches long. The flexor carpi radialis muscle lies directly under the incision; this I push aside with the handle of the scalpel; Dr. McMann now holds it aside with retractors. I find the outer border of the flexor sublimis digitorum muscle and lift it inward with my finger. The lower border of the pronator radii teres comes into view, and the median nerve is seen emerging from beneath it. Two inches below the margin of the latter muscle we find a strong cicatricial band of muscular and connective tissue which crosses and presses deeply against the median nerve. Below the constricting band the sheath of the median nerve presents a normal appearance, while above this band the sheath is greatly enlarged; it bulges somewhat, and is nearly three times as large as below the point of constriction. The normal, glistening aspect of the nerve-sheath is lost for two and a half inches; the sheath looks as if it had been severely contused, and is in the condition of degeneration known as "cloudy swelling." I now split open the sheath for an inch; some of the nerve-filaments have been severed and are separated, others appear to be continuous. Inasmuch as the sheath is intact and some of the nerve-filaments appear to be continuous, it will be good surgery to close the sheath, trusting to the reparative powers of nature to restore function by the process

of regeneration. The sheath serves as a tube to guide the proliferating cells from the central to the peripheral nerve ends; there is, therefore, no call for nerve suture or the implantation of any foreign substance to promote repair. It is claimed that all foreign substances used to connect severed nerve-fibres, such as long-distance sutures, bone tubes, and nerve tissue, simply serve the purpose of a guide to direct the regenerating cells between the severed nerve ends.

I will now cut down on the musculo-spiral nerve in front of the external condyle of the humerus. As the head of the radius rests upon the front of the condyle, it will be more difficult to find the nerve than in a normal arm. Here it is partly clamped under the inner and back side of the head of the radius. Its sheath is stretched, considerably widened, and appears tense. I loosen it from its environments, thus relieving any pressure and stretching. The radial nerve can now be seen. We will pull on it to see if it is movable; it is evidently bound down somewhere in its course. I extend the incision downward along the inner margin of the supinator longus muscle three inches, trace the nerve in its course to the lower end of the incision, and find that it is bound down by adhesions. These are next torn and separated. As the parts supplied below the ulnar nerve are also paralyzed, it will be necessary to find the nerve-trunk deeply between the inner border of the flexor sublimis digitorum and the flexor carpi ulnaris muscles. Here we come in contact with a firm cicatricial band, similar to the one in relation with the median nerve, extending across the ulnar nerve. I sever this band; the nerve presents the same appearance as the median, but exaggerated.

Having liberated and inspected four nerve-trunks involved in the paralysis, we will remove the constrictor, proceed to control hemorrhage, and close the wounds. You notice there are only two superficial wounds, though four nerves were exposed. The incision over the ulnar nerve was made after lifting the skin from the line of incision over the median nerve and drawing it over the ulnar side of the arm; thus I avoided making a separate incision over it through the skin. As it is, there will be two long scars. Let me impress upon you the importance of mutilating tissues as little as is consistent with thorough work. Hemorrhage having been controlled, the wounds are now stitched, and iodoform powder is dusted over them. The arm is wrapped in a thick layer of gauze and cotton; it is placed in a right-angle splint, so as to secure perfect rest.

We expect gradual restoration of function. Sensation generally returns earlier than motion. How much restoration may be expected depends upon two factors,—namely, (*a*) the amount of damage done to the nerve-filaments; (*b*) the restorative power of nature. It will be a slow process. We shall doubtless find considerable improvement in this arm within a week or two. The rigid condition of the hand will give way to a soft, pliable feeling in a few days. Within a week sensation will be partially restored, and after two or three weeks I shall look for some power of motion, which improvement need not be expected at once. Before operating the patient was told that he must not look for great gain in less than from six to nine months' time, and he must not be too sanguine of the final result. It will be impossible to promise him anything definite as to that. We have a reasonable hope for restoration of function.

After the wounds heal he will be treated with electricity,—faradism over the muscular tissue, and the galvanic current over the congested and thickened nerve-sheaths. The patient will be presented before you from time to time.[1]

## GOITRE.

Mrs. B., aged fifty-nine years, presents herself for operation to-day with an unusually large, rapidly growing goitre. She was treated in this clinic last fall for Colles's fracture. Your attention was called to the goitre at that time. It was much smaller then than it is to-day. She was advised by me to have it removed upon her recovery from the effects of the fracture. The rapid increase in the size of the tumor during the past six weeks has frightened her, and she earnestly desires to have an operation done. The patient wanted me to promise to limit the operation to the larger growth on the left side. She wished to know if the operation was dangerous to life. Of course, when a patient asks you point blank what the risks of an operation are, it is your duty to give a frank reply. There is danger to be apprehended in this case. The growth is an unusually large and vascular one; there are many large veins running over the surface of the goitre; there are numerous accessory veins. We shall doubtless find a complete net-work of vessels all over the capsule of

[1] Seven months after operation sensation is completely restored; the patient can flex and extend his hand, and can touch the end of the first finger with the thumb. He also has free wrist motion.

FIG 2.—Before operation.

FIG. 3.—One week after operation.

the growth, and other adventitious vessels extending into the substance of the tumor. Observe the varicose condition of the veins in front of the sternum and along the root of the neck. The external and anterior jugular veins are enormously enlarged. The first great danger in this case will be from hemorrhage, and we have a normal salt solution prepared to meet this emergency in case of necessity.

Auscultation reveals the fact that the mitral valves are slightly roughened. A mitral murmur is recognized. There is a condition of arterio-sclerosis present, which adds a second element of danger to the operation, infinitely increasing the hazard of anæsthesia. A third risk consists in the possibility of air embolism. With every precaution that may be taken, there is always a possibility, where so many large veins are to be severed about the neck, that air may enter one of them and reach the heart, and such a contingency might lead to instant death. There is still another danger that always exists in the removal of a goitre,—namely, injury to the recurrent laryngeal nerve, causing paralysis of the larynx.

The patient has been thoroughly prepared for the operation. A cathartic was given her three nights ago,—hydrarg. mass, grains 10; aloes, grain 1,—followed the next morning by magnesium citrate. The neck was scrubbed day before yesterday; a compress of green soap was applied, and to-day the parts were again washed. We will now have her sit for a picture before you. There, we have taken two views. The neck is quickly covered. Our assistants will immediately cleanse it again and proceed with the anæsthetic. I make an oblique, slightly curved incision downward and inward, after Kocher, its concavity looking upward over the most prominent part of the larger growth towards the median line. This incision crosses the anterior jugular vein and many smaller veins. These vessels come plainly into view. We will tie each with two ligatures and cut between them. With all the precautions we take, the hemorrhage is very free, requiring the use of many hæmostatic forceps. We have laid bare the stylo-hyoid and sterno-thyroid muscles. These are drawn to one side with retractors, and the capsule of the goitre is exposed, literally covered with a net-work of irregular blood-vessels, chiefly veins. A transverse incision is made through the capsule; with the fingers and a sound the large mass is gradually enucleated. The blood wells up at a fearful rate; with hot water compresses the bleeding is controlled. In proceeding with the enuclea-

tion we must be prompt, deliberate, and watchful for hemorrhage. We have now removed the largest portion of the goitre. Next I enucleate the smaller growth from the median portion of the neck which rests upon the trachea The hemorrhage is even more profuse. Let us pack the cavity with sponges soaked in hot water. We now proceed to ligate the more important vessels; the smaller vessels contract nicely; there are, however, a number of arteries to tie. We have ligated eighteen, an unusually large number of ligatures for a goitre operation. This may properly be called a vasculo-parenchymatous goitre. In some portions it has undergone degeneration. Here and there we find large centres of colloid. There are great vascular spaces in the substance of the growth, and the parenchymatous tissue is much hypertrophied. The hemorrhage having been completely controlled, the wound will be closed with silkworm-gut sutures. A strand of sterilized gauze is inserted, to be retained in the wound for forty-eight hours. The patient will be brought before you a week from to-day.

This is the fifth goitre removed in this clinic during the past six months. Many goitres are a constant menace to the patient's life. We enucleated one from a man forty-eight years old. The growth was much smaller than this one. The patient, however, suffered more on account of the location of the goitre. It was lodged between the sternum and the trachea, and he was almost suffocated by it a number of times. His voice was stridulous and extremely weak. He could stand no exertion, and was in fact in imminent danger of losing his life by strangulation. The operation was a critical one. We had hardly gotten him anæsthetized when the pulse and respiration ceased. There was no time to be lost. I hastily ran the knife transversely across his neck, opened the capsule, and lifted the tumor forward, taking pressure off the trachea, while my assistants practised artificial respiration. Success rewarded our efforts. The patient made a prompt recovery, and walked about the hospital on the fourth day. He was discharged cured in two weeks.

Eight weeks ago I was summoned in great haste to see a girl eighteen years of age. The messenger said she could not get her breath. We had to ride two miles. Upon our arrival at the house the girl was dead. Death evidently was caused by suffocation. The tumor was wedged between the sternum and the trachea.

The ordinary, diffuse colloid goitres frequently yield promptly

to medical treatment. The thyroid extract is the most universally used to-day. I have treated three-fifths of my cases during the past four years with thyroid extract, and the others with iodine, and have found that the latter remedy accomplished a cure more rapidly than the former. I believe the iodine is the basis of the most successful internal treatment. A hard, parenchymatous goitre, as a rule, will not yield to medication, and therefore enucleation or excision is advisable, especially if the goitre threatens life, or manifests a malignant tendency in middle life, the first indication of which is rapid increase in growth.

# SCHROEDER'S OPERATION.

CLINICAL LECTURE DELIVERED AT TUFT'S COLLEGE MEDICAL SCHOOL.

BY CHARLES GREENE CUMSTON, B.M.S., M.D.,

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GENTLEMEN,—The patient whom I am about to show you this morning is thirty-two years of age. Her menses began at the age of fifteen. She has had four labors at term; the last one, eleven months ago, was difficult, and there was a partial retention of the placenta, for which she was curetted. Since this last labor the patient has suffered a good deal from a bearing-down feeling in the lower abdomen and some pain on the right side, which has weakened her considerably. There is an abundant leucorrhœa, and once or twice a month she has a metrorrhagia with clots.

Examination showed an enlarged uterus in physiological anteversion; the sound enters the uterine cavity nine and a quarter centimetres. There is a bilateral laceration of the cervix, with ectropion of the lips, which are covered with granulations and a number of cystic productions.

The diagnosis in this case is subinvolution of the uterus, endometritis of both the corpus uteri and the cervix, with laceration of the latter; and the treatment will be curettement followed by Schroeder's operation.

As to-day the student leaves his medical school with the idea that every gynæcological case is to be treated by a laparotomy, I am glad to take this opportunity to discuss a less radical but quite as effectual operation when rightly applied in a given case. I refer to Schroeder's operation, with which you should all be familiar, as you will often be called upon to perform it.

Subvaginal anaplastic amputation of the cervix by Schroeder's method is a conservative operation, which removes part of the cervix and reconstructs a new one composed of *healthy tissues*. If the operation has been performed under good conditions, the new cervix will have all the physiological attributes of a normal organ; it will be soft and elastic and will dilate perfectly at the time of labor, so that the pregnancy will be perfectly normal.

Let us now consider those cases in which Schroeder's operation is of value, and we will begin with metritis without extension of the inflammation to the adnexa. We often meet with patients who have a chronic metritis with laceration of the cervix, with ectropion and sclerocystic degeneration of its mucosa, as in the case I have shown you. This type of lesion resists all local treatment, such as applications of tincture of iodine, chromic acid, etc. Only slight catarrhal conditions will get well by such treatment.

When we are dealing with an inveterate type of metritis of the cervix, with its glandular hypertrophy and adenopathic granulations of the mucosa, nothing will cure until this diseased mucosa has been freely removed. The lesions are well marked in the cul-de-sacs of the glands situated deep down in the mucous membrane and muscular tissue, and cannot be reached by the curette, but by removing the diseased tissues by Schroeder's method a cure is quite certain to ensue.

When a cervix is small and its mucosa rosy in color, it is most infrequent to find the endometrium of the corpus diseased, but, if such is the case, you will find that a simple curettement will be all the treatment required. If, on the contrary, there is an inveterate cervical metritis, its very adherent mucosa cannot be removed by the sharp spoon; so, when you find a *large cervix* and an endometritis of the corpus, curette the cavity thoroughly and then do either an Emmet or a Schroeder operation.

Now, it has been demonstrated over and over again that, in order that a trachelorrhaphy may be efficacious, its performance is delicate and not easy. The complete removal of all diseased structures is necessary, and when freshening the parts we should cut well into the healthy tissues.

It is often quite difficult to recognize the point at which the diseased structures end,—that is to say, the cicatricial tissue,—and often one may well hesitate to remove large amounts of tissue the loss of

which may only be replaced by another process of granulation and cicatrization. In spite of a free excision a diseased mucosa may still remain in the cervical canal, while if amputation of the cervix be done the operation is easy and very sure to be radical.

Now, we have other cases in which there is a chronic cervical metritis with laceration and sclerocystic degeneration of the mucosa but without ectropion, and for such cases many gynæcologists perform trachelorrhaphy. But here again it is evident that all the diseased structures are not removed by the operation, and, in reality, we enclose a diseased mucosa within the cervix, which cannot be acted upon by local antiseptic treatment, so that, instead of improving the state of affairs, things are made worse. The patient will complain of pain, leucorrhœa and hemorrhage will continue, and finally an amputation of the cervix will be resorted to, which should have been done in the first place.

Again, we have a condition of cervical metritis with a sclerocystic change but without laceration or ectropion, the cervix being either large or small.

In the former case the patient will complain of abdominal pain, irregular menstruation, and more particularly of a severe leucorrhœa which resists all treatment. The cervix will be found enlarged but of normal aspect, excepting that it is of a dark-violet color.

Now, if you carefully examine such a cervix, you will see that, instead of having a perfectly smooth surface, the latter is slightly granular, and this condition is present in several degrees. The mucosa being inflamed, the follicles are interested in the process and their orifices become closed up; they then may undergo three kinds of changes, the first two of which correspond to the clinical type that I have already described:

They slowly attain the size of a millet-seed, and form smooth, round, elastic cysts filled with a viscid and sticky fluid. These are what we commonly call Nabothian cysts. The contents are often purulent. These glands do not undergo a complete development, but remain at the surface of the mucosa. The fundamental lesion being here the same as in the conditions above mentioned, amputation of the cervix is indicated.

In cases where the cervix is small, we are usually dealing with nulliparous females who present the ordinary symptoms of chronic metritis with marked, painful, and dysmenorrhœic phenomena. Ex-

amination will show a small and well-formed cervix without any laceration, and you will most likely believe that your patient is neuropathic, and a treatment will be instituted in consequence.

These cases, perhaps, are not generally well known, and usually a metritis is diagnosticated only when the cervix is enlarged and covered with granulations. The physician forgets that the patient is nulliparous and consequently the cervix is small. But, for all that, there is an infectious metritis, and the lesions have penetrated from the cervical mucosa to the deep glands. The disease becomes all the more chronic the smaller the external os, and in such case a bilateral incision with excision of the diseased tissues is distinctly indicated. Such a treatment not only does away with the metritis, but ends the sterility produced by the conicity of the cervix and the attending catarrhal condition.

Every time that the lesions are sufficiently pronounced to justify Schroeder's operation and when they predominate in the cervix, it seems to me that their clinical aspect may be placed in one or the other of the four types that I have described, but I am well aware that their symptoms may vary greatly in different cases.

We now come to the subject of metritis especially marked in the corpus uteri, a chronic metritis of the corpus with hypertrophy of the uterus, which is a frequent affection. Sometimes it is a multiparous female with phenomena of subinvolution, at other times we have a chronic infectious metritis, following or not acute symptoms, usually of the gonorrhœal type. The clinical picture is, however, the same, —viz., pain in the abdomen, difficulty in standing upright or walking, dysmenorrhœa, metrorrhagia, an abundant yellow, thick leucorrhœa, and all the ordinary reflex phenomena attending diseases of the uterus.

Local examination will reveal a *large* cervix, either with or without ulceration, while palpation shows an increase in the size of the uterus, its cavity measuring eight or nine centimetres instead of six or seven. A careful treatment—viz., curettement of the uterine cavity—is necessary, or involution will take place afterwards.

But very old cases are often exceedingly obstinate, and it is in just these cases that amputation of the cervix will bring about the desired result. In these cases of chronic metritis with congestion of the uterus, subinvolution, and hypertrophy of the parenchyma curettement is necessary, but at the same time some operation that will

hasten involution should also be added, and I believe that Schroeder's operation will do this, if I may judge from personal observation.

There is another affection of which I would say a few words, and that is hemorrhagic metritis of long standing which has not been influenced by curettement. It is evident that in many cases the metrorrhagia is due to a lesion of the ovaries and not to a diseased condition of the endometrium, and that castration is indicated; but I believe that, before attempting such a radical interference, amputation of the cervix should first be tried, and if not successful we may then resort to abdominal section.

It is not so long since inflammation of the adnexa was considered a contraindication for curettement and, *a fortiori*, for any operation on the cervix, but at the present time it may be said that any operation on the uterus having for its end a disinfection of the organ is without gravity, and you are all aware that, even when acute symptoms are present, a careful disinfection of the vulvo-vaginal canal and curettement of the uterus are followed by a fall in the temperature and improvement of the general condition of the patient.

I have on several occasions curetted women with a slight salpingitis, with the result that the enlarged and distended tube disappeared a few days after the operation, resulting in a complete cure, and some of them had been advised to undergo a laparotomy by other surgeons. Trélat pointed out long ago that salpingitis could be cured by simply treating the endometritis.

It is, of course, impossible to say just at what time a diseased tube can be influenced by a cure of the existing metritis,—that is to say, in other words, the removal of the focus of infection which has produced the lesions in the adnexa. Now, it is not to be doubted that a Schroeder's operation on the cervix following a curettement will do very much in hastening a cure, because of the phenomena of involution that it produces not only in the uterus but in the adnexa as well. By these two operations we may often save the ovaries in young women and restore them to perfect health.

I will now say a few words regarding plastic operations on the cervix to correct malformations and of amputation for conditions other than metritis, and we will first of all discuss the advantages and disadvantages of Hegar's and Simon's operations.

Hegar's operation consists in a section of the cervix, the mucous membrane being sutured around the external orifice. The sutures

are passed through the mucous membrane of the vagina and are brought out through the external os, which, when the operation is completed, is lined by the mucosa. The cervix can be cut in different ways, so that almost any shape may be given to the organ according to the indications of a given case. In some cases the sutures are placed around the entire circumference of the cervix so that the vaginal mucous membrane is united with that of the cervical canal, while in others the vaginal mucosa of the sides is united with the vaginal mucosa and, at the middle line alone, the cervical mucous membrane is sutured to that of the vagina.

When the tissues have a normal consistency, Hegar's suture will give excellent results, even when the cut surface is not excavated; but, if the cervix is hard and rigid, it is not an easy matter to depress the borders of the wound so as to bring them together in good coaptation. Under such circumstances the tissues will form a hernia between the sutures; the stitches often cut the mucosa, especially when they simply include the latter without passing through a layer of muscular tissue.

The method of Simon is as follows: The cervix is split up on both sides to the vaginal cul-de-sacs. The incision of the upper lip is carried from the internal mucosa into the tissues obliquely from below upward; the other incision in the same lip, starting from the external mucosa, is carried so as to join the first and intercepts a conical segment whose base is below. The two flaps thus obtained are sutured together and the same technique is followed in the posterior lip. Then each corner is brought together by two or three sutures.

This is most surely an easy operation to perform and has its indications, but in order to be successful the mucosa of the cervical canal must be perfectly normal, a condition of very infrequent occurrence, even in cases of malformation and others where symptoms of metritis are *nil* or very slight. Simon's operation, which is applicable in all cases where the cervical canal is sufficiently resistant, will often fail when the tissues of the cervix are thin, as is observed in certain malformations of the vaginal portion with dilatation of the cervical canal.

The applications of Schroeder's operation are, on the contrary, more general, and the only circumstance which contraindicates it, and which for that matter is infrequent, is found in those cases where the vaginal mucosa covering the external aspect of the cervix is in-

vaded by a neoplastic or inflammatory process, because enough tissue cannot be found to make a good flap. Schroeder's operation is more difficult to perform, but, when well done, it has the following advantages over the other two methods,—namely, it gives the cervix its normal shape; there is no open surface left at the bottom of the vagina, and primary union is secured if proper care be taken in regard to asepsis; the amount of cicatricial tissue is very small; and, lastly, by lining the cervical canal with healthy mucous membrane, we avoid infection and its resulting metritis in the future, a feature that often compromises the results in the other methods.

The indications for Schroeder's operation in other cases than metritis are several. First we have congenital or acquired malformations involving the entire cervix or simply one of its lips, such as exaggerated conicity or hypertrophy of either the anterior or the posterior lip, with or without ectropion of the external os. Inveterate stenosis of the external os or cervical canal which has resisted other methods of treatment may be cured by this operation. Secondly, anteflexion or anteversion of the uterus, with pain, sterility, metritis, and dysmenorrhœa, are cured by this operation, on account of the changes produced in the static conditions of the organ as well as by the involution induced. Hypertrophic elongation of the cervix is the third indication for Schroeder's operation, whether or not accompanied by prolapsus, and whether due to subinvolution, to chronic inflammation, or to a congenital condition. Thirdly, we have *benign* neoplasms of the cervix, such as myomata, polypi of the cervical mucosa, non-malignant papillomata, etc. But, gentlemen, when you have a carcinomatous growth of the cervix, even although it may be limited to only a very small portion of the organ, Schroeder's or any other amputation is *absolutely contraindicated*, and a vaginal hysterectomy is then the only operation by which you may hope radically to cure your patient, and the question of cancer leads me to the consideration of other contraindications of this operation.

All pelvic foci of suppuration are contraindications, but remember what I said relative to salpingitis. A uterus well bound down by adhesions, which cannot be drawn down to the vulva, is not in a condition contraindicating Schroeder's operation, for the very reason that there is almost always a chronic metritis which is difficult to cure by other treatment.

In order that we may obtain a successful result from this opera-

tion, there are two conditions which must be attained: first, in cases of chronic cervical metritis, which is the fundamental indication for this operation, the diseased mucosa and underlying stroma must be freely removed, so that the flaps will be composed of only healthy tissue; and, secondly, these flaps must be well and evenly cut, in order to secure primary union and leave no raw surface at the bottom of the vagina.

The usual preparations for gynæcological operations in general are used here. The pubis is shaven, the vagina scrubbed with soap and water and then irrigated with creolin, lysol, or a one-to-one-thousand solution of nitrate of silver. An assistant then depresses the fourchette with a broad valve, and the cervix is seized with a pair of forceps and drawn down to the vulva. Never neglect curetting the uterus first, as a preliminary step of the operation, and, when this is done, we commence the amputation, the patient being placed in the dorsal position.

The first step of the operation is a bilateral incision of the cervix, accomplished by inserting one blade of a scissors into the cervical canal and then cutting upward to the insertion of the vagina, which should be done by one clip of the scissors. (Fig. 1.) Another clip of the scissors on the other side divides the cervix into two halves, an

FIG. 1.

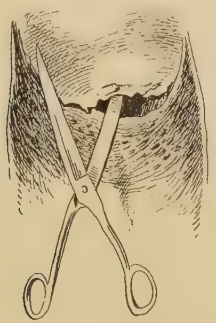
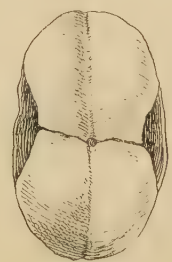


FIG. 2.



upper and a lower one, and by spreading them apart we have the entire cervical canal under the eye (Fig. 2), and consequently the extent of the lesions can be seen, and at the same time we can judge how much tissue must be removed.

The second step of the operation is the removal of a cuneiform segment, with its base at the lower part of the flap, which should in-

clude the mucosa of the cervix and at least two-thirds of the thickness of the lip. This is done with the knife by making two incisions, one being transverse (A B, Fig. 3), the other longitudinal (A C B,

Fig. 3.

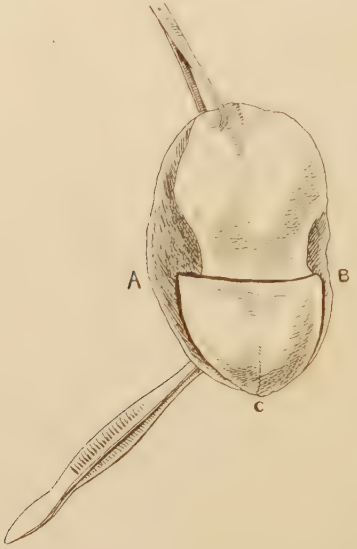


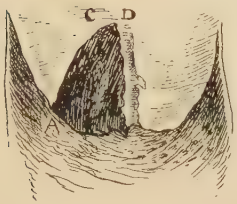

Fig. 3). An assistant holds the upper flap upward with a pair of bullet-forceps, while the operator seizes the lower lip with a dissecting-forceps and makes an incision from one side to the other (A B), going down into the tissues to the extent of two-thirds the thickness of the flap. This incision limits the base of the cuneiform segment to be removed. Then, beginning at B (Fig. 3), the knife is brought down along the right border of the flap to C, and the incision is continued to A, and thus the segment is outlined. It is then removed by scissors. Some little blood is lost, but the best hæmostatic is the sutures, and if a small artery bleeds freely it may be clamped and tied with catgut. I must again repeat that the segment removed should include at least two-thirds of the thickness of the flap, because on this point rests the ultimate success of the operation. Fig. 4 shows a badly made incision, while Fig. 5 shows the correct manner of making it. I need hardly say that a cuneiform segment is removed from both the upper and lower flaps. The result of a properly performed Schroeder's amputation is shown in Fig. 6.

The third step is the suture of the flaps, which should be done

with a full-curved needle. The needle is first passed through the apex of the flap, and then, directing it forward, it is pushed through the middle of the slice of tissue formed by the transverse incision and is brought out through the cervical canal; one suture in the middle and one on each side are usually quite enough; then they are tied. The same sutures are passed in the other flap, only in the

Fig. 5.

Fig. 4.



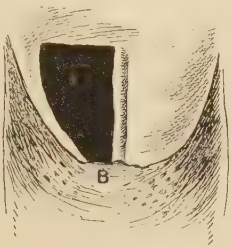
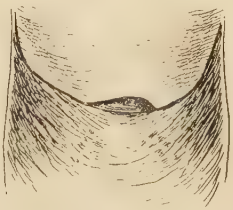




Fig. 6.



opposite direction. All that now remains is to suture the lateral discission, which is more difficult.

An assistant pulls on the sutures of both flaps, which, remember, must not be cut, drawing them to one side so as to bring the lateral discission into view. Before uniting the borders they should be evened off, if necessary, and then they are sutured, beginning at the upper angle, two or three sutures being usually enough.

The borders should be brought together perfectly, and especial care must be taken if any bleeding occurs at the upper angle of the wound. The result is seen in profile in Fig. 7, while the entire cervix in full view is represented by Fig. 8. A final result which does not have the appearance here given is a bad one.

Fig. 7.

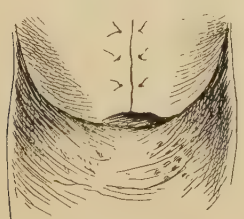
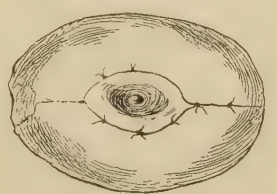


Fig 8.



The operation being completed, a strip or two of carbolate of bismuth gauze is carefully packed around the cervix, after irrigation of the vagina, and a pad is placed over the vulva. The dressings

should be changed on the third day, the vagina irrigated, and more gauze packed in around the cervix. The dressings should be changed with every aseptic precaution; otherwise you may infect the stitches and suppuration will result. If the dressings are rapidly soaked by the discharges, they must be changed oftener than every three days.

The sutures may be removed on the twelfth day, and the patient may be allowed out of bed on the next; but if, as sometimes happens, pain is felt in the pelvis, rest in bed for at least three weeks is indicated.

In closing this lecture I would say a few words regarding the examination of the cicatrix in order *to prevent stenosis of the cervix.* There are cases on record where a stricture of the cervical canal has occurred after Schroeder's operation, but I believe that this accident is due to a badly performed operation. Consequently, before the patient returns to her home, it is well always to pass a uterine sound, in order to be sure that the cervical canal is patent.

# CHOLECYSTOTOMY; NEURECTOMY; THORACOPLASTY.

CLINICAL LECTURE DELIVERED AT THE COLLEGE OF PHYSICIANS AND SURGEONS.

BY WM. M. HARSHA, M.D.,

Professor of Operative and Clinical Surgery in the College of Physicians and Surgeons, University of Illinois, Chicago, Illinois.

GENTLEMEN,—The first case presented to you was operated upon for the removal of gall-stones from the gall-bladder two weeks since. He is a skilled mechanic, twenty-eight years old, married, presenting a good family and personal history, and is of good habits. Four years ago he suffered the first attack of biliary colic. The pain was intense, sudden in onset, accompanied with vomiting, and lasted a few hours, stopping suddenly, and leaving marked soreness in the region of the gall-bladder for several days. Since then he has had repeated attacks of a similar character, requiring strong anodynes, and usually lasting but a few hours. He has not had jaundice, which may not be present if the common duct is not obstructed for approximately twenty-four hours. The frequent recurrence resulted in great loss of time, much pain, and some impairment of health. Under an anæsthetic no tumor could be found. No search was made for stones that may have passed. The character of the pain caused by the passage of the stones would indicate in a general way the nature of the stone, owing to whether facets are present or absent.

There was no history of any great rise of temperature other than what might occur from a temporary disturbance.

The dangers of perforation, peritonitis, hepatic abscess, and continued disability determined the operation.

On opening the abdomen by a vertical incision the gall-bladder readily presented in the wound, showing normal size and color, except as the yellow stones dotting its surface could be seen through its walls. The bladder was distended with them. It was stitched to the

peritoneum, incised, and sixty-eight stones were removed, varying in size from a small hickory-nut to a grain of wheat. As many as one thousand or more have been reported in a single case, but the more usual number is eight or ten. Search was made in the duct and no stones found therein. The presence of pus in the gall-bladder, besides the febrile symptoms which it produces, imparts to the organ a yellow color entirely different from the normal dark liver color. The finger is best for detecting stones, while after the incision has been made the forceps and small scoop aid in removing them. A tube was left in the bladder and the ordinary copious sterile dressing applied. Recovery was rapid, the operation and recovery being attended with less pain than a single attack of colic would produce. The tube was left in four days, and considerable bile escaped. Since the patient has been walking about, a little further leakage is present. The wound, as you now see, is nearly closed, and will probably be entirely healed within a week or two.

The ideal operation—*i.e.*, closing the gall-bladder with fine catgut or silk, and then the abdominal wound—might have been done, as there was no pus, but we cannot always be sure there are no small stones in the ducts. In this case two or three small ones came away on removal of the tube.

Case II.—The next patient presented was operated on last Wednesday for spasmodic neuralgia of the inferior dental variety (tic douloureux). He is fifty-five years of age, by occupation a carpenter, of good antecedents and personal history. This was his second operation; the first was done six years ago for the same trouble and in the same nerve. You see that he has had the teeth extracted on this side with the hope of relief. There are many causes named for this most painful affection. Carious teeth, affections of the ear, brain syphilis or tumor, empyema of the frontal sinus, exostosis producing pressure on the nerve, malaria, reflexes, neuritis from cold, rheumatism, or other causes. In this case there is a history of injury by a falling hammer, sixteen years ago, on the lower jaw, at the point of exit of the inferior dental nerve. Within a year he began to have pain, which recurred more frequently and increased in severity. Six years ago he was operated upon through a long curved incision, the scar of which you see. The cure lasted until one year ago. Assuming that a resection was done in the usual place a little above the angle of the jaw, I cut down on the sigmoid notch, and, having no trephine small

enough, drilled small holes through the jaw in the shape of a horse-shoe, thus deepening the notch to a point where the nerve enters the bone. The nerve was twisted by means of artery-forceps from both directions, probably an inch or more being removed. Care was taken to avoid wounds of branches of the facial nerve and Stenson's duct. The incision was made in the line of the old scar. There has been no pain since the operation, and the fact that pain was confined to the inferior dental nerve and its distribution offers the hope of a permanent cure, although that cannot be assured. If the neuritis, which is often present in these cases, has not extended beyond the reach of this resection, his chances are good. Should the pain recur, as is often the case, in from two to five years, the nerve must be attacked higher up, even as far as the Gasserian ganglion perhaps. The excision of the scar, with the removal of such portions of the stump of the nerve as may be reached, would probably extend the immunity from pain some years, should it recur.

Constitutional treatment by tonics and cod-liver oil is indicated.

Case III.—The next patient presents himself for Estlander's operation, or thoracoplasty. He is a carpenter, twenty-four years of age, of good family and personal history up to the beginning of his present trouble. Two years ago he contracted pleurisy with serous effusion, was aspirated twice, after which the effusion became purulent. He was then referred to me for operation by Professor Quine, in his absence from the city.

Three pints of pus were evacuated through an ample incision at the site of the present opening which you see on the right side. At that time there was very slight expectoration, and in the sputum tubercle-bacilli were found. He was treated with systemic remedies, and, beginning the day after the operation, the pleural cavity was carefully irrigated with iodine; carbolic or sublimate solutions being used daily for several weeks. He improved in general health, the tubercle-bacilli disappeared from the sputum, his weight increased, and he resumed light work; but the sinus refused to heal, although the amount of pus diminished until only one or two drachms were evacuated each day. In addition to the ordinary anti-tubercular remedies he has had injections of Paquin's serum. He improved somewhat while taking this combined treatment for two months.

We make a vertical incision, beginning three or four ribs above the present opening, baring first the rib next to it, peel off the peri-

osteum, and with sharp cutting bone-forceps cut away about three inches of bone. We now find a large cavity extending in every direction, and the right lung contracted to one-quarter its normal size. The lung cannot expand to fill the chest and the chest wall cannot fall in to fit the lung. We proceed with one rib after another until five or six are excised, from one to four inches of each rib being cut away. We now, with scissors, cut away the pleura, which is as thick as sole-leather, together with the periosteum, to prevent other bone formation, and so can bring together the cut edges of the thorax. We find considerable granulation tissue, which is scraped out with a sharp spoon. The bleeding from the divided intercostal arteries is stopped by pinching them—sometimes a ligature is needed; they are easier dealt with if peeled off with the periosteum and left until the ribs are cut away.

The cavity is now irrigated, emptied of all clots, shreds, and débris, a large double drainage-tube laid in, the edges of the wound approximated and partially sutured, and a copious antiseptic dressing applied. The patient will have one-fortieth of a grain of strychnine by hypodermic injection and heat applied when in bed to prevent shock, which is severe in some cases of this kind.

The prognosis is not the best, owing to the tubercular history. We see, from the size of the cavity, that without operation there would be no hope of the wound closing. There is probably very little tubercular trouble in the lungs, judging from the slight expectoration and the absence of temperature and of tubercle-bacilli.

The prognosis in early tuberculous cases is better than was formerly believed. The fact that healed or latent tubercular lesions have been found in so many bodies after death from other causes (estimated by some at thirty or forty per cent.) teaches us that very many recover, or do not succumb to the disease when present. The prophylaxis of tuberculosis (most pleuritic cases are such) requires that measures be taken to isolate or educate patients with the disease; and in the well, to keep the physiological resistance up to par, thus preventing colds, which invite infection of the nasal, pharyngeal, and bronchial mucous membrane.

# Gynæcology and Obstetrics.

## THE VALUE OF EXPRESSION IN CASES OF MODERATE CONTRACTION OF THE PELVIC INLET.

CLINICAL LECTURE DELIVERED AT THE CHICAGO POLICLINIC.

BY C. S. BACON, M.D.,

Professor of Obstetrics in the Chicago Policlinic; President of the Obstetric Staff of the Chicago Health Department, etc., Chicago, Illinois.

Gentlemen,—To-day I will call your attention to a procedure that is not new, that is often quite important, and at the same time much less employed than its value merits. I wish to speak of expression of the fœtus, and particularly of pressure applied to the fœtal head in cases of hindrance to labor at the pelvic inlet, caused by inefficient pains combined with a moderate degree of pelvic contraction. I will introduce the subject and illustrate the value of the procedure by the report of a case which recently occurred in private practice.

The patient was a woman thirty-five years old, with a bad family history and weak constitution, who had been married thirteen years. She had had three miscarriages during the first years of her marriage, and three years ago became pregnant for the fourth time. This pregnancy progressed normally, and at term the labor began by premature rupture of the membranes twelve hours before regular uterine contractions were felt. The physician previously engaged was called at the beginning of labor and remained with her in constant attendance during its progress. After the labor had lasted about thirty-four hours, during which time, according to the report of the patient, the pains were strong, she became convinced that the fœtal motions, which previously had been very marked, had ceased. Thereupon she called the attention of her physician to the fact, and

then active measures were taken to deliver the child, which had not yet entered the pelvis. After the very severe application of high forceps a dead child of at least average size was delivered. The puerperium was disturbed by a chill on the third day and fever lasting till the twelfth day. The patient remained in bed five weeks.

Here I wish to digress from the report of the case in order to comment on the management of this labor. The physician was in attendance from the first, yet no exact measurements were made to determine the size of the pelvis, even after the labor had progressed for hours without engagement of the head. No attempt was made to listen to the fœtal heart-tones. These were two grave mistakes. When a physician assumes the responsibility of conducting a case of labor, he should exercise reasonable skill in determining the presence or absence of pathological conditions. The physician is without excuse if, either through ignorance or negligence, he fails to determine the size of the pelvis. Also, when he takes charge of a case of labor, he has two lives intrusted to his care,—the mother's and the child's. It is his business to keep himself informed of the condition of each of his patients. He can know the condition of the child by observing the fœtal heart-tones quite as well as he can the condition of the mother. If, through ignorance or negligence, he overlooks the danger which confronts the child and allows the child to die, and remains in ignorance of its death for hours afterwards, he is entitled to the same blame that we would attach to him were he sitting by the bedside of a patient with typhoid fever or pneumonia and be for hours ignorant of the fact of the death of such a patient. I must speak very strongly in condemnation of such mistakes as were made in this case. You could have no excuse that would justify you in the eyes of friends or relatives of a patient who should die in your presence, and be dead for hours before you knew of the fact. Just so you must hold yourselves guilty if you allow the fœtus to die and remain in ignorance of its death. So many physicians who come here to the Policlinic tell me that they have never heard the fœtal heart-tones, that I feel it my duty to insist very strongly upon the importance of listening to the heart-tones in each case, and in this way keeping track of the condition of the child.

The patient first consulted me on the 24th of July, 1896. She had been told by her family medical attendant that she could never have a living child. She came to consult me regarding her general

condition. She suffered from constant headache, weakness, nervousness, and irritation of the bladder. She also wished my opinion concerning the possibility of bearing a living child, although she was disposed to believe the opinion before given and greatly feared the dangers of labor.

I found her anæmic; blood, Fleischl 68; the chest normal, except a slight cog-wheel inspiration over the left apex; heart normal; urine, specific gravity 1.013, alkaline, without albumen or sugar.

The pelvic measurements, which chiefly interest us, were as follows:

| | |
|---|---|
| Spines | 25 |
| Crest | 28 |
| Baudelocque | 18.5 |
| Conjugate diagonalis | 10 |
| Conjugata vera, estimated | 8 5 |

I felt my responsibility in giving an opinion concerning the possibility of her having a living child. Both the patient and her husband, who was a well-to-do merchant, were very anxious for a family. The measurements showed the pelvis to belong to the class of moderately contracted pelves. The fact that a fair-sized child had been extracted by forceps, without perforation, led me to the opinion that she could be delivered of a living child, and I so informed her. Relying upon my judgment, the patient became pregnant some months later. Her pregnancy was carefully watched, progressed quite favorably for six or seven months, when, on account of the serious and fatal illness of her father, she became very much exhausted, and at term was in poor condition to face the ordeal which she looked forward to with great fear.

You may imagine with what interest I watched the labor which began on the 8th of August. Regular pains began about 6 P.M. On my arrival at 8 P.M. I found the pains fairly strong, the os dilated to admit only one finger, the head still movable above the inlet of the pelvis. At eleven o'clock the membranes ruptured, while the cervix was dilated only to admit four fingers. For the next hour the pains were strong and well assisted by the efforts of the patient. Nevertheless, there was no progress in the engagement of the head. Considering the experience of the first labor and the size of the pelvis, and the condition of the patient, I became convinced that it was unjustifiable to wait longer for the unaided efforts of nature. Then

came the important question what to do. There were just three possibilities, forceps, turning and extraction, and expression, with perhaps the fourth eventual possibility,—symphyseotomy.

You have heard me state the objections to the forceps in these cases. The name "high forceps" is applied to an operation when the forceps are employed before the head has entered the pelvis. While the application of the forceps to the head in the pelvis after rotation is often a very easy operation, "high forceps" in cases of contracted pelves is one of the most dangerous of all obstetric operations, both to the mother and child. We can easily see the reason for objections to the high forceps when you consider how the head must be grasped and extracted. The head must descend through the inlet with its smallest diameter, corresponding to the smallest diameter of the inlet, —that is, the biparietal diameter of the head corresponding to the antero-posterior diameter of the inlet. When I apply the forceps to the unengaged head, you will see that the blades must grasp the head in its long diameter,—that is, one blade is applied to the face and the other to the occiput. Compression of the blades must increase the biparietal diameter of the head. This diameter was already larger than the antero-posterior diameter of the pelvis, and this compression increases the disproportion. Consequently, the increased and excessive amount of traction necessary, resulting in the possibility of much injury to the child and much bruising to the soft parts of the mother, must follow.

These theoretical objections to the high forceps, which are easily demonstrated on the pelvis or model, are abundantly sustained by statistics. It is now universally recognized that high forceps is a serious operation, and one attended with a high mortality both for mother and child.

Another objection to forceps in this case was the incomplete opening of the cervix. Through the insufficiently dilated cervix the forceps must be introduced; the result is very likely to be a deep tear extending perhaps to the attachments at the junction of the cervix with the uterus, leading to serious hemorrhage, or perhaps rupture of the uterus.

Version and extraction, if carefully done, will be a much safer operation for the mother than the forceps operation. For the child, on the other hand, delay in the extraction of the head will lead to very grave danger, and is to be avoided, if possible.

We shall be very fortunate if we can avoid both of these grave operations. This I succeeded in doing by the procedure before mentioned. It was carried out in this way: The patient was anæsthetized with ether, then placed in the Walcher position. You know what the Walcher position is and its advantages. Dr. Walcher made the discovery, almost accidentally, that when a patient is in the extended position, with the thighs extended on the pelvis, the conjugata vera is considerably longer than when the patient is in the extremely flexed lithotomy position. This is due to the fact that the sacro-iliac articulation is not a synchondrosis, as was formerly taught, but that it is a true joint. The movement is on a transverse axis located in the second sacral vertebra. When the thighs are extended, the sacrum rotates on its axis, and the promontory is brought farther away from the symphysis. The difference in the length of the conjugata vera between the position of extreme extension and extreme flexion is about eight millimetres; thus when the patient is in the Walcher position the conjugata vera is about eight millimetres longer than when she lies in the lithotomy position.

You see the extreme importance of this discovery in the management of this class of cases. Not only in expression, but in all obstetric operations where the head has not yet entered the pelvis, the Walcher position should be used. It is easily employed by putting the patient across the bed, with the hips at the edge and allowing the thighs to fall. In this case, after the patient was anæsthetized and put in the Walcher position, expression was begun. Pressure was applied directly to the head, taking account of the direction of the axis of the inlet. The somewhat thin abdominal walls allowed the head to be exactly felt, and pressure was applied to the occiput, the forehead, and the anterior side of the head, at times, by means of the ends of the fingers and thumbs of both hands, and again with the fists closed by the row of extensor surfaces of the first phalanges, protecting the abdomen from the knuckles with a towel. In less than five minutes the head pressed into the pelvis. The anæsthetic was then withdrawn, and the further progress of labor was left to nature. In about two hours the cervix was fully dilated and the head at the pelvic outlet. At this time the patient became somewhat exhausted; pains were weak; the fetal heart-tones, which at the beginning of labor were one hundred and thirty-four per minute, began to increase, reaching one hundred and sixty; and, in order to avoid any possible

danger to the child, the forceps at the outlet were applied after an ineffectual attempt at expression by pressure on the fundus of the uterus. The child was born alive and vigorous; weighed seven and a quarter pounds; was fifty-two centimetres long, and had the following measurements of circumferences of the head:

| | |
|---|---|
| S. O. B. | 32.3 |
| S. O. F. | 33.5 |
| O. F. | 37 |
| O. M. | 40.5 |

The subsequent history of the case up to the present time has been quite uneventful. The mother has had no fever and the child is thriving vigorously. You can easily understand how much the proper management of this case meant to this family. Considering the age of the mother, and her sad experience with the first labor, the death of this child would have meant a childless family. Had I pursued the waiting policy for several hours, until the mother had become exhausted and the uterine pains had ceased, and when the child became in imminent danger of asphyxia, and then resorted to the high forceps, we should in all probability have had a dead child, an injured mother, and a hopeless future for the family. From my observation, I am inclined to think that the latter procedure is the usual one in such cases, and hence I relate this case as a strong argument of the value of expression.

Expression is not a new procedure; it is probably one of the oldest of obstetric operations. We know that among the semibarbarous people it is often the custom for the midwives, or those who assist women in labor, to press on the uterus to aid in the expulsion of the child. This manipulation was resorted to hundreds of years ago. Since the development of scientific obstetrics, however, expression, as a method of operation for delivery, has been much neglected. The greater certainty of the various methods of applying traction,—namely, extraction and the use of the forceps,—has caused the simpler method to be quite generally overlooked. It was not until some time after Credé had developed his most valuable method of expressing the placenta that the expression of the fœtus was developed into a scientific method.

In 1867, Kristeller elaborated the procedure and gave quite broad indications for its use. His object was to expel the contents of

the uterus by pressing upon its fundus. This pressure acted in two ways: It excited increased uterine pains, like other kinds of massage. It also directly provided an expulsive force, acting in the same way and supplementing the abdominal pressure. The method described by Kristeller consisted, after removing the interposed bowel, in grasping the fundus of the uterus with both hands, with the fingers behind and thumbs in front, and pressing downward in the axis of the obstetric canal, at first gradually, then with more and more force, ten to fifteen seconds, then gradually relaxing. These movements are to be repeated at suitable intervals of one to five minutes. In a case of sensitive abdomen or uterus an anæsthetic is given. If the uterus lies too much to one side, it is carried into the median line.

The conditions and indications for the application of Kristeller's method are clear and simple. Kristeller himself gives very broad indications. He believed the method was applicable in the first stage of labor before rupture of the membranes. It was indicated in all cases of delayed labor due to inefficient uterine contractions. A fairly wide pelvis is necessary for its employment. It was applicable in either head or breech presentations, but not in cross presentations. These indications are too broad and the conditions are not accurately enough laid down. Before the rupture of the membranes, the Kristeller method, by which I mean expression where the pressure is applied to the fundus, is not of any value. It is a valuable procedure in those cases which are not infrequent, and often very important where, after a long labor, the head rests against the perineum and cannot be expelled by the inefficient uterine pains or the feeble abdominal pressure. These cases are not uncommon in older primipara as well as in multipara with relaxed abdominal walls. After the publication of this method of Kristeller, it was quite in vogue in Germany and England, but later it seems to have been considerably neglected. One very rarely sees it used in a systematic manner in the European obstetric hospitals. In this country I believe it is very little taught and practised.

The procedure I have described to-night was published by Hofmeier in 1881. This differs, as you readily see, from the Kristeller method in the fact that the pressure is applied directly to the head; so it is designed to be used, especially in cases of contracted pelves, or in cases of disproportion in the size of the pelvic inlet to the child's head. It is, of course, applicable only in cases of moderate degree of

contraction. In a pelvis with a conjugata vera of less than eight centimetres, with an average size head, it would probably be useless; but in those cases of moderate contraction, between eight and ten centimetres, which are most important cases, and often they are most difficult to manage, it may, as in the case cited, be of infinite value.

One important question remains to be considered. What are the dangers of expression? First to the child. By the Hofmeier method a case is reported where the lip of a child was considerably bruised by the pressure applied to the face. No serious cases, however, have been reported, and it is not likely that any serious injury can occur to the child from pressure so applied. A possible danger from the Kristeller expression has been suggested,—namely, separation of the placenta. Bidder reports two cases where the child died during the application of expression, which result he thinks was due to separation of the placenta and consequent asphyxia of the child. For the mother, premature separation of the placenta might lead to hemorrhage, but no such cases are on record so far as I know. By the Hofmeier method, which I have described to-night, there is a possible danger of rupture of the uterus. As has been pointed out by Bandl, rupture of the uterus generally occurs in the lower uterine segment. In cases such as you have all seen, where the lower uterine segment is very much thinned out as a result of a long labor, the uterine wall may become very much attenuated; and in such a case it is quite conceivable that pressure applied by the fingers to the head would cause a rupture. This danger is especially to be thought of, for cases where expression is indicated are just the ones where distention of the lower uterine segment takes place. It is possible to avoid the danger to some extent by placing two or three thicknesses of cloth or towel between the abdominal wall and fingers. It is only necessary to keep in mind the possibility of danger from this source.

# ETHYL BROMIDE IN OPERATIONS FOR HEMORRHOIDS; ECTOPIC GESTATION; ANTEFLEXION; REMOVAL OF THE UTERUS FOR MALIGNANT DISEASE.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON HOSPITAL.

BY E. E. MONTGOMERY, M.D.,

Professor of Gynæcology in the Jefferson Medical College; Gynæcologist to Jefferson and St. Joseph's Hospitals; Ex-President of the Philadelphia Obstetrical Society; Ex-President of the Pennsylvania State Medical Society, etc.

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Gentlemen,—This patient is now undergoing the administration of ethyl bromide. It is quite interesting that you should witness the administration of this drug, as it is quite evanescent in action, and is a very efficient anæsthetic for short operations. This woman underwent an operation on January 12 for the removal of the uterus. She had suffered from hemorrhages, and so much distress and discomfort that, with the hemorrhagic condition and the size of the uterus, I felt it better to extirpate the organ. As a result of the pressure of the large uterus upon the rectum, the circulation of the anus had been obstructed, producing hemorrhoids. I did not wish to complicate a vaginal hysterectomy, however, by treatment of hemorrhoids at the same time. I show you the temperature record, which immediately after the operation shows a temperature of 96° F., but she soon reacted. The highest temperature was 100° on the tenth day. The temperature has been nearly normal for several days. I bring her here to-day for the purpose of relieving the hemorrhoids. They have greatly decreased in size since the operation, but, as there is considerable eversion of the rectal mucous membrane, it is better they should be treated. We have cleansed the vagina simply to have the parts free from discharge. For inspection of the rectum, or rather the lower part of it, the anus, no instrument is better than a Sims's speculum. It enables us, by turning it about, to examine the entire under surface of the rectum and thoroughly study the structures.

The largest hemorrhoid is on the anterior surface. I draw out the mass with a hæmostat, apply a compressor to its base, and with the thermocautery proceed to burn off the external portion. I do this slowly to char the tissues in the grasp of the compressor, and thus insure the more thorough control of the bleeding. As I loosen the clamp I see there is no bleeding. This is a ready, effective, and quick way of treating hemorrhoids. There is a slough of the burned tissue which comes off, a cicatrix forms, contracts the mucous membrane and decreases the subsequent likelihood of its eversion. There is less danger and much less discomfort than there would be with the use of the ligature. It is important, however, to see that hemorrhage is controlled before the part is turned back, otherwise you may have bleeding occur from a vessel, which may be excessive, before there is any external discharge; in other words, a hemorrhage into the bowel. This patient will at once be given a grain of extract of opium by suppository. It is very well, often, to give in addition a suppository of five grains of iodoform. I am glad to show you this patient following the operation of vaginal hysterectomy. The plan of treatment I have just used would not be applicable where the hemorrhoids are situated externally. If there is a hard clot external to the sphincter known as external hemorrhoid, the best plan of procedure would be to incise the clot, turn it out, and prevent a tag forming, as is frequently seen after such hemorrhoids have recovered, the clot having become organized. Internal hemorrhoids have a tendency to cause a rolling out of the mucous membrane every time the bowels are evacuated, requiring the patient to make an effort to replace them. If the patient is much upon her feet, this same eversion takes place with a portion of mucous membrane in the grasp of the sphincter, giving rise to strangulation and exceedingly great distress.

*Ectopic Gestation.*—The next patient, twenty-three years of age, has father, mother, two brothers, and one sister living and in good health; puberty occurred at fourteen, the menses were regular, painless, and lasted six days. She has been married five years and has had two children. The labors were normal, and one miscarriage occurred on the 8th of February last. The last child was born in December, 1895. On November 28, 1896, the regular time for her period, the menstruation began, since which she has had constant discharge, severe pains dating back to November, none prior to that time; no headache, bowels regular, and very good appetite. In this patient we

have a history of a woman who has had two children, a miscarriage in February, and since November of the same year, following her menstruation, there has been a continuous bloody discharge, which still exists in February. In other words, for nearly three months there has been a bloody discharge. I have gone over the various causes of hemorrhage, conditions which produce it outside the uterus, outside the genital tract, conditions which occur as a result of disease of the uterus itself, and the changes which take place in its structure which may be a cause. The fact that this woman is twenty-three years of age, and that this hemorrhage has occurred for the last three months, would exclude from our minds, to a certain degree, the probability of it being malignant. You should not, however, be absolutely certain. We recognize that malignant diseases may occur as early as the twentieth year or even younger. You will remember, only a week ago I showed you a patient in this clinic but thirty-four years of age, in whom malignant disease was so marked as to render it questionable as to whether an operation could be done and afford any hope of radical cure. We have hemorrhage occur in other conditions. It takes place as a result of disease outside the uterus, in the pelvic structures, and in this patient there is at present, I am told, some enlargement external to that organ. I have not examined her, but will now do so to determine, if possible, the condition. She has pain in the left side. As the finger is introduced into the vagina, we find the uterus is situated in the median line, the os directed slightly forward, the entire uterus apparently raised forward, while posterior to it is a mass which rounds out and fills up Douglas's pouch, projecting from the left side, which is quite tense. As I press the finger upon the left fornix of the vagina, I notice a marked pulsation of the vessels. You see my finger is stained with blood. This pulsation of the vessels shows that there is pressure above, interfering with the return circulation, and the pressure renders more prominent the left uterine artery. This is an exceedingly interesting case. A woman twenty-three years of age, with two children, a history of hemorrhage continuing for three months, and upon physical examination a mass filling up Douglas's pouch, extending to the left side, a history of pain in this region, which came on suddenly at the menstrual period. So far as we can determine there is no special history of inflammatory condition; she has had continuous pain since, and no indication of the presence of inflammatory trouble which would have caused it.

A patient who has had a miscarriage during the year, which took place subsequent to a labor, with a sudden change from the ordinary menstruation to continuous discharge, with an enlarging mass in the pelvis, leads me to believe that we have an ectopic gestation which has ruptured into the broad ligament, filled it up and dissected beneath the peritoneum in this region. You can readily see that the pressure of such a mass would give rise to interference with the venous circulation; the uterus consequently is engorged, and the result is, we have a patient with a continuous uterine discharge. In such a history as this, we have to decide as to the course of treatment. Shall we permit her to go on, with the hope that nature will absorb this mass and the patient recover without any operative interference? In many cases it is possible this will take place, in which an ectopic gestation sac has ruptured, with accumulation of blood in the pelvis, the mass becomes encysted, partly absorbed, partly organized, and ultimately disappears. The blood-clot must undergo a process of organization; it is gradually absorbed with thickening of the part, an increased mass of exudation necessarily results. Such patients recover, but they are more or less crippled, as they have been exposed to a certain amount of danger from the presence of such an accumulation. A mass of this kind, accumulating in the pelvis in close proximity to the large intestine, may readily become infected by the passage of germs through the intestinal walls. These abscesses when they discharge are exceedingly offensive. There are different methods of treatment in such conditions. Formerly, the only method considered advisable was to open the abdomen, break up the mass, and turn out any accumulation of clots. Nature has generally already thrown out plastic exudation. When the material has escaped into the peritoneal cavity, we now, instead of opening through the peritoneum in chronic cases, prefer to incise the vagina, and in such a patient we make an incision through the posterior cul-de-sac. In that situation, we know there is but one-fourth to one-third of an inch in thickness between the posterior fornix of the vagina and the peritoneal cavity. The cul-de-sac is rendered still more prominent by the accumulation within it, and the posterior fornix of the vagina is so prominent that there is no danger in making an incision through it into the peritoneum. The only case in which there would be any possible danger would be in one in which the hemorrhage was recent, and in which the recent clot might readily be displaced, permitting a

recurrence of hemorrhage. Even in such cases, with a free incision posteriorly, the fundus of the uterus may be turned down, and with it the ovary and tube dragged into the vagina, where the pedicle can be ligated and hemorrhage controlled. One advantage of this procedure is that we do not interfere with any barriers nature has already provided against the infection of the general peritoneum. We do not, consequently, infect the general peritoneum with the broken-down material. Another advantage is, that we have at the most dependent portion of the pelvis a free opening in the sac, from which the material can be readily removed, through which irrigation may be practised, and in which we may subsequently thoroughly pack gauze to insure the maintenance of the opening and the escape of any material which we are unable to remove. The drainage is at the most dependent portion of the pelvis, with gravity rather than against it, as we would have if we opened the peritoneal cavity through the abdomen. And, in addition to this, the patient is free from the effects of an abdominal incision; from the pain, distress, and discomfort consequent upon it. She is saved, too, from the possibility of ventral hernia, which may sometimes take place where drainage has been necessary. In a patient in whom hemorrhage is recent, and consequently where there may be more difficulty in managing the case, it is better to do an abdominal incision, so that the bleeding vessel can be at once secured. In this patient the result is a fortunate one compared to what we sometimes find. We may have, at once, a history of severe collapse; the patient falling in a faint, passing from that into another, and, finally, death taking place in a few hours, or even an hour after the first symptoms. I remember a case which came into the out-patient department a few years ago, where we examined the abdomen, recognized a mass upon the right side, and could feel pulsation at its base, as from a vessel as large as a radial artery. The patient had a retroverted uterus. I had examined her and left the room, was called back in ten minutes to find her lying on the sofa, face blanched, covered with cold perspiration, pulseless, and with every symptom of violent internal hemorrhage. I at once urged the necessity for an operation, but she would not consent until her husband could be secured. I sent a carriage for him and within an hour had him present, in the meantime using stimulants to maintain life. We opened the abdomen, found a quart of free blood in the peritoneal cavity, secured the bleeding vessels, but the patient soon died,

hemorrhage having continued so long that she could not recover from the shock. Had we been able to secure an immediate operation, I am confident we would have saved her life. It is just as important in these cases of internal hemorrhage to secure the bleeding vessel as it would be in a case of hemorrhage of the femoral or radial artery.

*Anteflexion.*—The next patient is twenty-one years of age, single, a dressmaker, whose parents, four brothers, and two sisters are in good health. Puberty occurred at fourteen; menstruation was regular, and lasts, as a rule, from one to two days, but never very free. The flow is very painful. During the past twelve years she has complained of constant pain in the right ovarian region, gradually getting worse, not always during the menstrual epoch. This pain is aggravated by exercise, and she has constant leucorrhœa, headache, and constipation. The appetite is good. Examination of this patient will be attended with more or less pain; we will give an anæsthetic, so that we may be able to eliminate the element of pain and nervousness. The essential features in the history of this patient are the severe pain during menstruation, the leucorrhœal discharge immediately following it, and the pain in the right ovarian region, which is unaffected by the menses. After the administration of the anæsthetic, using bromide of ethyl, I introduce the finger into the vagina and find the uterus is slightly anteflexed; the anteflexion takes place between the internal and external os. There is a distinct turning forward of the cervix. I find no enlargement on the right side, nor is there, in so far as I can determine, any apparent tubal involvement upon either side. To determine still further the condition, I insert a finger into the rectum, and passing it over the fundus, find no enlargement or thickening of the broad ligament on either side. This patient undoubtedly has inflammation of the uterus and endometritis associated with anteflexion. The pain and distress in the right ovarian region of which she complains are evidently neurotic. I do not think there is any special disease of the right ovary which should occasion it. Formerly, it was the rule in such patients, where they complained of continuous pain in one or both ovaries, that oöphorectomy should be done; but experience has disclosed the fact that they have been but little improved by such an operation. If the patient had both ovaries removed, and still has pain, she is very likely to condemn the operator; we are consequently very slow to subject a patient to operation unless pathological conditions can be determined

by physical examination. These patients who suffer from such neurotic symptoms are the most unsatisfactory ones to treat. This patient has inflammation of the uterus, as is evident by the discharge from that organ. Such an inflammation would be difficult to remedy so long as the anteflexion exists. The proper plan of procedure, then, would be to dilate and curette the uterus, in addition split through the posterior lip towards the posterior fornix of the vagina, and stitch the mucous membrane of the cervical canal to that of the vagina to prevent a contraction of the parts, which would take place if such measures were not practised. This plan is beneficial in these patients, not only relieving the dysmenorrhœa which occurs as a result of the swelling up and formation of a valve, but interfering with the escape of the discharge until she practically goes into labor to force it out. In addition, the operation renders these patients much more likely to become pregnant. One of the frequent causes of sterility in women is anteflexion, which interferes with the probability of conception, not only through the valve-like action of the canal, interfering with the entrance and the contact of the spermatozoa with the ovum, but also through the fact that the spermatozoa is deposited in the posterior cul-de-sac, behind the orifice of the uterus or external os, and is not so likely to enter the canal and cause conception. The splitting of the cervix will have no special influence on the process of gestation, nor on that of parturition. Under aseptic precautions, with the method suggested, we have but little cicatricial tissue, which consequently does not interfere with the normal development and dilatation of the cervix.

I have seen, to-day, a patient of so interesting a character as to be well worthy of being brought to your attention, a woman who had undergone her ninth confinement on Thursday last; as the labor was rather prolonged, instruments were applied, and delivery was secured without much difficulty. The placenta was completely removed, and everything seemed perfectly favorable. During the time of her delivery, however, a mass was noticed which seemed to be situated high up in the pelvis, whose character we were unable to determine. When I saw her yesterday evening she had a temperature of 103° F., a severe chill, and all the indications of septic processes. The uterus had been curetted, and nothing found in the cavity of the organ to indicate there was putrid intoxication. In other words, no clots or portions of placenta or membrane were

retained, which by its decomposition produced ptomaines and their absorption, and what is known as putrid intoxication. The question was whether we had to deal with sepsis, or whether, notwithstanding the care exercised by the very capable attending physician, some infection had resulted, leading to sepsis. Placing my hand over the abdomen, I discovered a mass nearly as large as a child's head situated in front of the uterus, with the pedicle apparently not larger than my finger. I believed it to be a fibroid growth of a subperitoneal variety, which had been extruded from the uterus. I suggested to the physician, as he had found no history of the condition indicating putrid intoxication, that it was possible that the circulation of this mass had been so interfered with as to cause its loss of vitality, and the elevation of temperature was the first symptom of resulting inflammation. We decided to await results, have the uterus again irrigated night and morning, and see what would take place. I saw her this morning, and found her again in a severe chill; passing the hand over the mass, I could distinctly feel crepitation, a sensation as if adhesions were forming. The neck of this tumor felt very much as if it had been twisted; there was a long pedicle. I have no doubt it has been twisted; so I propose to open the abdomen and remove not only the tumor but the uterus as well.

*Removal of the Uterus for Malignant Disease.*—You remember a week ago I showed you a patient thirty-four years of age, with a mass projecting into the vagina, into which I told you I could introduce my finger. The finger was passed into the uterus, the tissue was apparently friable. On examination under an anæsthetic this tissue broke down. You see here at the edge of the vagina, where it is attached to the uterus, a mass of tissue that extends down to the vaginal surface. In this patient we could have done nothing by vaginal operation, because there was not sufficient firm tissue to afford a secure hold. In addition to that, the vaginal operation could not have been practised because of the enlarged uterus, and the distance we would have had to go upon the broad ligament would have endangered the ureter, so I made an abdominal incision. After opening the abdomen, we ligated the upper part of the broad ligament, which secured the ovarian artery on either side; then split the peritoneum over each common iliac at its bifurcation into the internal and external, and ligated on either side the internal iliac. In doing this we push off with the peritoneum each ureter. Having ligated these

arteries, we proceed at once to dissect off the bladder anteriorly, and cut through the broad ligaments on either side without waiting to ligate any vessels. The only bleeding was from the veins. I opened through the pelvis into the vagina and removed the vaginal orifice in contact with the tumor, so that in this way we were outside of the disease through its entire extent, except on the left side, where a mass extended into the broad ligament, in the aim to remove which we opened into the bladder. This I at once sewed up, and upon examining it I found I had included the ureter in the suture, so it had to be reopened, and, dissecting the ureter out, it was turned into the bladder and the opening closed. Having done this and introduced sutures in such a way as to control bleeding from the veins, the cavity was sponged, the peritoneum sewn up, gauze packed into the vagina, filling up the pelvis, and this covered over with a fold of peritoneum from the bladder. In that way all the drainage was outside the peritoneal cavity. The patient stood the operation fairly well. In the after part of the day she had a temperature reaching 104° F. This was evidently reaction temperature, as it soon subsided. She complained of considerable distress and discomfort in the region of the heart, rapid pulse, and bleeding.

Note.—This patient did fairly well for ten days, when she had an attack of tetanus from which she died. As to the origin of the tetanus, we were unable to determine whether it could have been infection from some catgut ligature which was used in ligation, or from some other cause which we had no means of determining. The ligature, however, had been thoroughly boiled prior to its use.

The case of sepsis referred to in this lecture was operated upon later in the day, when the mass which was supposed to be a fibroid growth was found to be a dermoid cyst, the pedicle of which had been twisted off and the mass was attached to a fold of peritoneum below, which was continuous with the upper surface of the bladder, and above to adhesions of the omentum. This fold of peritoneum at the end of the tumor had evidently been injured during the delivery, as a large clot was poured out beneath the peritoneum which extended down the side of the vagina nearly to the vulva. This clot was partially decomposed, and it was evidently due to its presence that the putrid intoxication of the patient had occurred. The tumor and uterus were removed; the patient, however, did not manifest much improvement in temperature, and died about thirty hours later.

# Ophthalmology.

## SQUINT AND PALSIES OF THE OCULAR MUSCLES.

CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA POLYCLINIC.

BY EDWARD JACKSON, M.D., Denver, Colorado,

Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine.

Gentlemen,—The diagnosis of squint does not end with the recognition of the disease, but begins with it. The first question that you may ask yourselves is *whether squint is present.* I have spoken to you of the connection of diplopia with squint; and how you would recognize such diplopia by its disappearance when one eye was covered; how you would thus separate diplopia due to deviation of an eye from diplopia due to other causes.

By covering the eye and watching its deviation you also separate apparent deviation of the eye due to an eccentric position of the cornea from real deviation of the eye. If both eyes are properly directed towards the point looked at without excessive effort on the part of some of the ocular muscles, when one eye is covered no change will occur in the position of either eye, both eyes being already turned towards the object fixed. But if one eye is turned towards the object fixed and the other is turned in some other direction, upon covering first one eye and then the other, certain changes of position occur.

You tell the patient to look at a certain point, then place the hand in front of the fixing eye, and he being prevented from looking at that point with the previously fixing eye, will look with the eye that has deviated, and will turn the deviating eye in the direction of the point he was told to fix. That turning of the eye that was before deviating is spoken of as the "recovery" of its proper position. The

eye that is covered, however, at this same time deviates. You have the squint transferred from what was the "fixing eye" when both were uncovered to what was then the "deviating eye." The "deviating eye" has become the "fixing eye," and the eye that normally fixed has become the "deviating eye." That is what happens when the fixing eye is covered.

If you remove the hand now from the fixing eye it will again fix in most cases,—that is, it will again be turned towards the point to which the patient's attention is directed. This eye having "deviated" while it was covered, makes the movement of "recovery" as soon as the hand is taken away; and the one which was the fixing eye when the other was covered becomes the deviating eye as soon as the other has again "fixed." The movement of deviation occurs in one eye and at the same time that of recovery occurs in the other. These movements occur when the hand is placed before the fixing eye or removed from the fixing eye. If you take the eyes in their usual position and cover the deviating eye, no movement will occur. The other eye will continue to fix and the covered eye will continue to deviate. In this way you can determine with certainty that there is a squint. You will remember that when squint is not present, but only apparently present by reason of the position of the cornea, no such movements occur on covering either eye.

The next question that you should ask yourselves with reference to any such case is, *whether the squint is concomitant or paralytic;* for these are the two great divisions of strabismus.

Concomitant squint consists in a false relation of the visual axes to each other. The ocular movements have a definite relation in squint which may be as constant as the relation they have normally; but in squint it is an improper relation. For example, if an attempt is made to look at a near object, in concomitant squint both eyes turn in, as both normal eyes turn in. But in the case of convergent squint, where the visual axes were already converged, they converge still more. When a distant object is looked at, they tend towards a parallel position, but remain still somewhat convergent.

In contrast with this, paralytic squint is not any false relation between the visual axes of the two eyes or any false co-ordination of the movements of the two eyes; but it is a failure of movement on the part of some particular muscle, failure of movement in some particular direction. Suppose, for example, we have paralytic squint

due to paralysis of the external rectus muscle, rather the most common form of paralytic squint. Suppose it to be the external rectus muscle of the right eye that is paralyzed or weakened, then the squint will only appear with those movements of the eye that call for action of the right external rectus muscle, the paralyzed muscle. In looking to the left, upward, downward, or at a near object the eyes will move properly together. But on looking to the right and at a distance, the right eye, on account of the weakness of the external muscle, fails to turn out properly. The left eye is normally turned. The left eye properly fixes, but the right eye deviates. This is the difference between concomitant and paralytic squint. It is the point which should be next investigated after the recognition of the existence of squint.

In practice this is done by causing the patient to look in different directions. Commonly we hold up a pencil or a finger before the patient and move it in different directions, telling the patient to hold the head rigidly directed forward, and not raise or lower it, but to follow the movement of the finger or pencil by turning the eyes. In a case of concomitant squint the two eyes will be seen to turn together, in all directions. But in a case of paralytic squint, in such movements as require the contraction of the paralyzed muscle, the eye it acts upon is found to lag behind the other. Thus, in paralysis of the right external rectus, the eyes move together in other directions, but on carrying the pencil to the right the sound eye follows it accurately, but the impaired eye lags behind, squint develops, and the farther it is carried to the right the greater the degree of squint. Instead of being the same when the eyes are turned in different directions as concomitant squint, the squint only begins when the paralyzed muscle is brought into action, and increases the more the paralyzed muscle is called upon to exert itself. Much of the time paralytic squint is entirely absent; and when it does exist, its amount depends upon the ocular movements attempted. The more the patient looks towards the affected side the greater the degree of squint.

If the squint is quite small in amount, particularly paralytic squint, the recognition of its nature may be better accomplished by investigating the diplopia caused than by watching ocular movements. Although a muscle may not be entirely paralyzed, its weakness may cause very annoying diplopia. In such cases the move-

ments of the affected eye do not stop short; the eye moves but a little less under the same stimulus. Under these circumstances it is difficult by mere inspection of the eye to be sure that such a deviation really occurs. But as soon as it occurs the patient begins to see double, and you get the indication of the squint by this diplopia.

If the squint is small in amount, it may not be possible to tell by inspection what kind it is. It may not be possible to tell whether the right eye is lagging behind when the eyes are turned to the right, causing a convergent squint due to paralysis of the external rectus muscle of the right eye; or whether the double vision is caused by the left eye lagging behind, owing to a weakness of the internal rectus muscle of the left eye, causing a divergent squint. Hence, in studying the diplopia, unless the failure of movement and consequent deviation is very marked, it is necessary to find out whether the right image belongs to the right eye or to the left,—that is, whether the images are *homonymous* or whether they are *crossed.*

This brings us to the third question to be asked in a case of squint,—viz., *What is the direction of the deviation?* In all cases of marked squint this third question will be answered with the first. In cases of slight paralytic squint it often requires a good deal of special testing to make sure of the answer. Whether the squint is concomitant or paralytic, the determination may be made by the diplopia. If it is a convergent squint, you have crossed images; if divergent, you have homonymous images. To determine whether the images are crossed or homonymous, you find out which image belongs to the right and which to the left.

One way is by covering the eye and asking the patient which image disappears. In using this method it is necessary to be on your guard against a certain source of error. You remember that when you cover the fixing eye, the eye that was before deviating immediately begins to fix, so that one may think the false image is the one that has disappeared, when really the false image has moved to the position of the true image, and the true image is the one that has disappeared. Take a case of partial paralysis of the external rectus muscle of the right eye. You have the patient look somewhat to the right so that the diplopia begins. If you cover the left eye, which is the fixing eye in this case, the right eye is immediately turned so that it fixes, and so quickly that the patient may think the true image

has remained, when really it has disappeared and been replaced so quickly that he has not noticed the change, and thinks that the false image has disappeared. On this account, simply covering the eye and asking the patient which image disappears is not always satisfactory. If, however, you cover one eye with a colored glass, so that the patient has both images still to look at, he can tell you which of the two images is colored, and you can then be absolutely sure which belongs to the right eye and which to the left.

While the method of covering with the hand is very convenient, and usually quite satisfactory, in some cases the patient will be misled, and will give a false answer, so that in using it you must be on your guard against that source of error. If by simple inspection of the eye you could see the kind of squint, the patient's mistake about the images would not mislead you, but in a case of very slight squint the patient's answer would be very liable to mislead you.

From this point the consideration of concomitant and paralytic squint diverges. In matters of pathology they differ entirely. Their causation is different, their course is different, their treatment is entirely different. As to the actual deviation and the connection of the squint with diplopia, they are identical, but beyond that they are entirely separate. Often they are treated in separate chapters in treatises of ophthalmology.

*Causes of Squint.*—Taking up *concomitant squint* first, its causes lie in the direction of false development in the visual organs and false co-ordination of movements. The best recognized cause for squint is an error of refraction, ametropia. We have convergent squint connected with hyperopia, and divergent connected with myopia. Another common cause of squint is deficient vision in one eye so that binocular diplopia is rendered impossible; and in that way the movements of the eye lose their normal guide to accurate co-ordination. This guide consists in the sensation of single vision so long as the movement is normal; and if such movement is departed from, double vision. Now, if double vision does not occur when false movements are made by one of the eyes, this guidance is lost; and false movements, deviations of greater or less extent, occur.

It has long been recognized that extensive opacities of the cornea are very liable to cause squint, that a blind eye usually deviates, and we know, since the ophthalmoscope has been used, many causes of blindness lie back of the iris. A certain number of cases occur in

which the eye fails of its normal development; we find sometimes coloboma of the optic nerve; instances of failure to develop vision in certain parts of the retina, which are probably of the same nature, but with the lesion far back in the nerve-tracts and not visible with the ophthalmoscope. Any of these causes of blindness in one eye may result in a concomitant squint.

On the other hand, paralytic squint arises from the ordinary causes of nerve lesions, causes such as affect peripheral nerves elsewhere. Neuritis is a cause of paralytic squint. It may arise in connection with rheumatism, or so-called cold, although such cases are not very common. A much more common cause of paralysis of the ocular nerves is syphilis, through the pressure on the nerve-trunks usually of gummata somewhere about the base of the brain. The ocular muscles are more apt than any other muscles in the body to reveal the presence of gummata, because of the long path at the base of the brain of the nerves supplying them and the great distance that they are exposed to the pressure from such causes.

Another cause of such palsies is found in the degenerations affecting the cerebro-spinal centres. The centres in the medulla for the ocular movements are, some of them, particularly liable to degeneration, so that in a large proportion of cases of degenerative disease of the nerve-centres we find the ocular muscles affected early. Paralytic squint is a very important early symptom of tabes.

Of course, differences in causation and pathology lead naturally to the differences of treatment. Concomitant squint is treated by lenses, by prisms, by tenotomy to readjust the muscles of the eyeball so that they will produce their normal effect upon the globe, or by muscular advancement which has the same purpose as tenotomy, and by orthoptic exercises.

On the other hand, paralytic squint is treated by the methods that we employ for paralyses of other nerves. Some cases are due to "cold" or to injury. Persons receiving severe blows on the head, or falling considerable distances, are liable to paralytic squint, probably due to traumatism of the nerve-trunks. These cases tend to recover spontaneously, running the course of any other paralysis from the same cause, in the course of some weeks. The recovery from more than a perfectly temporary functional impairment always requires some weeks to complete it,—time enough for the regeneration of the nerve-fibres. The treatment of such a case would be simply to avoid

during that period anything that might cause the squint to become permanent.

In the case of syphilis, the treatment, of course, would be the ordinary specific treatment, and for these cases the essential thing is potassium iodide, started with perhaps ten grains to a dose, and increased a grain, or two or three grains, every day, until the drug produces some of its physiological effects, or the paralysis is rapidly improving. The dose is only to be limited by one of those occurrences, the patient often taking half an ounce to an ounce a day.

I have taken up paralytic squint first, because in a certain number of cases it is a factor in the establishment of concomitant squint. While paralytic and concomitant squint are different as regards pathology and treatment, there is a tendency for them to come together at a late stage. A case of high concomitant squint will very generally lead to the actual weakening of the stretched muscle. In convergent concomitant squint there will generally be actual weakening of the external rectus muscle, the muscle that would have to be paralyzed to cause convergent paralytic squint, so that the concomitant convergent squint becomes in some measure paralytic squint at this stage. On the other hand, squint originally due to paralysis of the nerves supplying certain muscles, if it exists very long, develops into a partly concomitant squint. In the case of paralysis of the external rectus of the right eye, the eyes are converged at first only when they are turned far to the right. Presently the convergence occurs when the patient looks at all to the right of the median line. Later there may be excessive convergence when looking directly ahead; and still later the squint may become constant. This is particularly noticeable in paralysis of the oculomotor nerve.

If the paralysis is absolute, you have on one side of the eyeball a muscle with its normal power; on the other side nothing to oppose it, except the elastic tension of a muscle that is no longer used. The nutrition of the latter is imperfect, and it is undergoing degeneration. Such a muscle will not hold the eye against the elastic tension of the healthy muscle on the other side; so that there occurs a secondary contraction of the muscle that retains its power, and stretching of the paralyzed muscle, which causes the eye in the case of late paralytic squint to deviate constantly. The squint, from being originally paralytic, passes over to concomitant. In these cases you have a mixed form of squint, partly paralytic and partly concomitant. It is likely

that many cases of concomitant squint are essentially of this nature; not all, but a considerable proportion of them. Their occurrence should always be borne in mind.

Most patients with concomitant squint come with a history that the squint appeared in early childhood, and almost always with the statement that it followed some special occurrence, as after a fall or during or after a spell of sickness. In a large proportion of cases the only significance to be attached to such statements is that people try to explain everything they see. When a child's eye turns in they try to form some explanation of the occurrence and grasp at anything that occurred previously as a possible cause, so that very often the history you get of previous disease or injury does not amount to anything. But there are certain cases in which a slight, perhaps temporary, weakness or palsy of one of the ocular muscles, or more than one of them, has developed, and become fixed as a concomitant squint. The child has had, perhaps, an attack of diphtheria so mild as not to be noticed; but such attacks often are followed by weakness in some of the ocular muscles that has continued for several weeks, or even for months, as diphtheritic paralysis does. The paralyzed muscle has become permanently stretched, the eye in that way has been turned and caused to deviate, and the patient has become accustomed to using the eyes with one of them deviating. Now from such an origin you will have concomitant squint that may last as long as the patient lives. Occasionally one of these squints may be outgrown after several months or years, but probably some of the cases that come to us in later years as concomitant squint have originally started in the weakness or absolute paralysis of some particular ocular muscle.

The connection of squint with ametropia is twofold. That of myopia and divergent squint is quite different from that of hyperopia with convergent squint. The connection of hyperopia with convergent squint has been made too much of. When Donders called attention to it, it was not recognized that nine out of every ten eyes were hyperopic. We have a large number of cases of convergent squint in which the occurrence of convergence and hyperopia is merely a coincidence; one is not to be regarded as a cause of the other.

But there is a certain class of cases that I am sure are generally or always caused by hyperopia. They are cases of intermittent convergent squint, or of variable convergent squint, where the variations in

the amount of squint are very great, and the squint coincident with high hyperopia. We see a great many cases afterwards constant, in which the squint continues intermittent for weeks or months at first; and these are often of this nature. We see a few cases in which the squint continues to be intermittent throughout life, only occurring when certain efforts are made to use the eye; and these cases are invariably of this nature.

Such squint comes about through the excessive effort of accommodation necessitated by the hyperopia, leading to an excessive convergence. Now I do not think that this is due to a rigid connection between accommodation and convergence; that with each diopter of accommodation you must have just a certain amount of convergence. But the connection is more in the nature of an overflow impulse. You know that if you exert any muscle strongly you will find that the related muscles are also caused to contract. We cannot direct a very strong impulse to one muscle without affecting other muscles. We know that convergence and accommodation are very closely related physiologically. On that account, when the accommodation has to be very strong, it is always accompanied with a tendency to increased convergence; and in a good many cases this increased convergence actually occurs, and we have the squint occurring whenever the excessive accommodation is used. In these cases the squint is due to the excessive accommodation, and it is corrected entirely by correcting the hyperopia.

In a large class of cases the hyperopia probably has some share in the squint; it causes it to be variable in amount. If the squint actually occurs, after one eye has deviated you may lose the guide of double vision, so that it will make very little difference whether the eyes converge more or less; and in such a case, if there is hyperopia present, whenever the eyes are exerted for distinct vision the excessive convergence also will occur, and the squint will be greatly increased. In such a case a large part of the squint that is present is due to the hyperopia; and by correcting the hyperopia that much of the squint is cured. So it is of importance to give correcting glasses, and have them worn for some time before attempting to correct by operation what squint remains. If you attempt operative correction before that, you will probably overdo it.

In myopia the connection between the squint and the ametropia is anatomical rather than physiological. It depends upon the shape

of the eyeball and the shape of the orbits. You have the orbits as irregular conical cavities with their axes divergent, their inner walls being parallel. The myopic eye is elongated, and on that account is turned with greater difficulty in its socket. Convergence becomes more difficult. As soon as it becomes too difficult to be sustained for near work that is required of the eye (and, of course, the need of it is greater because the work has to be brought very close to the eyes) the more the eye is allowed to deviate outward. In most cases the eye at first simply does not turn in enough,—there is a failure of convergence. But as the case goes on and binocular vision is given up, the eye is allowed to deviate more and more until it settles in the orbit with its long axis in the position of rest, and you have the divergent squint, which almost invariably accompanies high myopia.

There being this anatomical cause for the presence of the squint, the proper measure of treatment is the wearing of correcting glasses, so that less convergence is required; and particularly the wearing of glasses to stop the progress of a myopia before it becomes high enough to cause an actual squint. After the squint has occurred, if it is of high degree, it becomes very difficult to correct it, even by tenotomy of the external, or advancement of the internal, rectus muscle. If you operate for such a squint you ought always to try to get an excessive correction at first; and you should combine with the operation on the muscles the change in the relation of the eyeball to the capsule enclosing it. Try to get adhesions of the capsule far forward on the nasal side, and allow the capsule to fall back on the temporal side of the eye. Cut off, not only the tendon itself, but cut freely around it, so that all the tissues related to the tendon may fall back. If possible, get an over-correction, because you will have to encounter afterwards this strong tendency of the eyes to continue to diverge.

# IRITIS.

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The iris is that part of the eye that gives it color, some being blue, some gray, others brown, etc. It hangs like a curtain in the aqueous chamber of the eye; it divides the chamber into two portions, the anterior and posterior. The iris, ciliary body, and the choroid are parts of the tract known as the uvea; the iris is the anterior part of this tract, and one of the most important structures of the eye. It is perforated at or near its centre, usually a little to the nasal side of the centre, by what is known as the pupil, and its posterior surface rests partly against the anterior capsule of the crystalline lens. It is attached to the sclera near the sclero-corneal junction by what is known as the ligamentum pectinatum; it is also attached to the ciliary body, as I have already stated, it being part of the uvea.

This iris, or membrane, which hangs or is suspended in the aqueous chamber, acts as a diaphragm, shutting off all peripheral rays of light, and allowing only the central rays to enter the eye, thus making vision more perfect. Suppose the pupillary opening in the iris were sufficiently large to admit the light through every part of the lens, what would be the result? We would have the rays of light passing through the periphery of the lens not focussed, which would produce a blurred vision. This can be easily demonstrated by experimenting with a microscope or other instrument of precision, which must be so adjusted as to admit only the central rays of light. The iris is made up of muscular fibres, principally circular in character, especially about the edge of the pupil, but there are also radiating fibres, although some authors claim that it is simply elastic tissue. It is enough for our present purpose to say that the frame-work of the iris is fibrous tissue, radiating and circular muscular fibres; in addi-

tion to this it contains blood-vessels and nerves (motor, sensory, and sympathetic), as well as pigment, which gives it color. This is about the anatomy of the iris so far as it need be recalled at present; its location and attachment give the angle, which is of considerable importance, between the cornea and iris, known as the angle of filtration.

Inflammation of the iris alone must be regarded as of considerable consequence, but it is almost impossible for the iris to become involved without the ciliary body and the choroid also becoming so to a greater or less degree, although we have many cases of iritis without any *apparent* inflammation of the ciliary body and choroid. The retina and optic nerve may also be involved in this inflammatory process without any involvement of the ciliary body or choroid, but this is unusual.

This subject of iritis is extremely important, because of serious mistakes that may be made in the diagnosis. A great many cases of iritis have been diagnosticated conjunctivitis, keratitis, etc.

In classifying iritis we can do so as to its cause; for instance, syphilitic, malarial, gonorrhœal, and rheumatic. Again, we have traumatic iritis, but this can be classified under the head of plastic—or when there is septic invasion, suppurative—iritis. Again, iritis can be classified pathologically, as serous, plastic, and parenchymatous, the suppurative again being a later stage of the parenchymatous or the traumatic with infection from without. There are some symptoms common to all forms of iritis differing in degree in the different forms: exudation of lymph, pain, fixed pupil (in serous iritis dilated or normal in size, and small in the plastic and parenchymatous forms), sclero-corneal congestion, photophobia, decrease of vision, etc., are such symptoms. I think the main diagnostic difference being in the cause, the amount of lymph and lymphoid cells exuded, and where deposited. For instance, in the serous form of inflammation the lymph is principally thrown out into the anterior chamber and deposited upon the posterior surface of the cornea and in the bottom of the chamber; sometimes the spots on the posterior surface of the cornea are so small as to be seen only by magnifying them very much. In the plastic form of iritis this lymph is deposited on the anterior and posterior surfaces of the iris and at the pupillary edge. In the parenchymatous form also lymph and lymphoid cells are exuded, but principally into the substance or parenchyma of the iris.

thus thickening the membrane very much and roughening its surface; one form frequently runs into another as serous iritis often becomes plastic also, but in the above I have given the main pathological differences.

Serous iritis has as its most common causes rheumatism and gonorrhœa, but either of these troubles may produce the sero-plastic or the plastic form of iritis. When gonorrhœal both eyes are more liable to be involved at the same time. The symptoms are pain, which is not so great as in the other forms of iritis; blurring of vision; circumcorneal congestion; pupil normal in size or dilated, but usually fixed; deposits of lymph in the lower part of the anterior chamber and on the posterior surface of the cornea, with probably increase of tension. The ciliary region is liable to be involved in this form of iritis and cause increased tension. In gonorrhœal or rheumatic iritis some of the larger joints are liable to be involved, but not necessarily so, though they are in a majority of the cases. Until recently it was thought that these joint-involvements came from the presence of gonococci in the joints, and Noyes, page 445, says, "and the same explanation is probable for iritis." I am sure more recent investigations demonstrate beyond any doubt that it depends upon toxines.

In plastic iritis the symptoms are more acute and more severe; more lymph is thrown out, and synechia posterior is more liable to occur than in the serous form; there is sometimes more swelling of the ocular conjunctiva, which may conceal the sclero-corneal zone. As I said above, the pain, loss of vision, congestion of the sclera, and discoloration of the iris are all more acute and more marked in the plastic iritis than in the serous. Again, the history of trauma or syphilis will assist you in making your diagnosis.

There is in the parenchymatous form of iritis still more severe symptoms. In addition to those symptoms seen in plastic iritis, the iris is much thickened, spongy, and mushy looking; it soon loses its normal appearance, and has in it new blood-vessels; the pupil is more liable to become occluded with organized lymph. The increase of lymph, lymphoid cells, and generation of pus, which sinks into the bottom of the anterior chamber and produces what is known as hypopyon, is one of the symptoms of parenchymatous iritis. It may be difficult occasionally to locate this pus, but if there is any doubt of its being in the anterior chamber have the patient lie first on one side

and then on the other, when if it is in the chamber it will change position, while if in the cornea it will not.

Now as to the diagnosis and differentiation of iritis from other inflammations of the eye: Suppose a patient with slightly blurred vision is inclined to avoid the light as much as possible, with some pain about the eye, worse at night than during the day, and we must remember in this connection that pain in all forms of iritis is worse at night. Have the patient face the bright light of a window, close the eye for a few minutes, then suddenly open it, and see if the pupil responds to light. As soon as a bright light strikes the normal eye the pupil contracts, and when you shade the eye from this light the pupil dilates; this is called the "play" of the pupil. If the other eye is not involved the two may be compared, closing the eye and then suddenly opening it, as I have indicated. Of course, in elderly persons the pupil is smaller than in the adult and in children. The pupil in iritis when exposed to the light does not contract, neither does it dilate when covered. The pupil in serous iritis may be normal in size or dilated, but there is no "play." There will be found a little sclero-corneal congestion; it must be remembered, however, that this form of congestion is also present in keratitis and other affections of the eye, and is not at all pathognomonic of iritis. We may have congestion of the globe in cyclitis, conjunctivitis, or with a foreign body in the eye. We must differentiate the sclero-corneal congestion from other forms, and this may best be done by remembering that it is deep-seated and not superficial: in the superficial congestion the blood-vessels are large and run in all directions, and they are movable by manipulation; by pressing upon them you can easily empty their contents, but they rapidly refill. In congestion of the deep blood-vessels one can empty them by pressure, but they are not movable, as they are in the superficial forms of congestion. The sclero-corneal congestion that we get in iritis is in a particular zone, deep seated; the vessels radiate outwardly from the cornea and are different in color from the others, being a little more purple, and, as I have said, can be emptied by pressing them with the lids, but they are immovable; if there is a keratitis, with or without an iritis, there will be an opacity of the cornea.

A man may present a history of rheumatism or of gonorrhœa; he may present partial or complete loss of vision; pain may be slight or intense; he has blurred vision, perhaps; he has the immovable pupil

that I have spoken of; he has the sclero-corneal zone. Upon examination flocculi are found in the aqueous humor and scattered about on the posterior surface of the cornea; there is a diseased iris, and, of course, the material it secretes will be diseased. Most of these cases have ciliary involvement; the lymph exuded is thrown out into the anterior chamber, and if the aqueous humor is tested it will be found to contain not only albumen, salt, and water, etc., the normal constituents of the humor, but it is filled with small flocculi which interfere very much with vision. If the lens, vitreous, and cornea are involved, this also interferes greatly with the vision. If lymph is thrown out into the pupillary field this, of course, produces blurred vision; loss of vision up to perception of light sometimes occurs from lymph thrown out into the field of the pupil. Pain results from engorgement of the internal tissues of the eye. The iris and ciliary body are filled with blood; lymph is thrown out, and by its pressure effects upon the nerves produces intense pain. Photophobia is a prominent symptom of iritis, the cardinal symptoms being pain, photophobia, sclero-corneal congestion, blurred vision, and synechia posterior. Of course, in iritis we have an increased amount of blood in the part, and the iris becomes very much thickened; we have the pigment layer disturbed and the aqueous muddy, which accounts for the changes which occur in the color of the eye in this disease. The blue iris becomes grayish; the black iris becomes a dirty, muddy brown. With the deposition of lymph and the increased amount of blood we have the iris thickened and its action interfered with, and unless we get hold of the case in time we have the iris completely tied down, producing what is known as posterior synechia. The difference between posterior and anterior synechia must be remembered: posterior synechia is where the posterior part of the iris and its pupillary edge is tied down to the anterior capsule of the lens; anterior synechia is where there is a perforation of the cornea, the iris falling forward, and as the aqueous humor escapes the iris comes in contact with the wound of the cornea and becomes adherent there. This attachment of the iris to the cornea is anterior synechia.

I have briefly outlined the most important and prominent symptoms of iritis, and now, how can the diagnosis be made beyond any doubt? I have mentioned the sclero-corneal congestion, photophobia, fixed pupil, discoloration of the iris, pain, blurred vision, posterior synechia, etc., and if there is any doubt about the iris being involved

it is the easiest thing in the world to make a diagnosis by means of the sulphate of atropia. Take one grain of the sulphate of atropia—or where it is desired to dilate the pupil quickly take two grains—to distilled water one-half ounce, put a drop of this into the eye three or four times in succession, and what do we expect to find? Unless the pupil is tied down, we will have prompt dilatation. Sometimes the pupil is completely tied down, and we can get absolutely no dilatation. In some cases where there are many adhesions we will get a saw-tooth or serrated dilatation, the pupil becoming dilated at points where adhesions do not exist or where dilatation breaks the adhesions partially. The pupil being adherent at some points to the anterior capsule, as demonstrated by means of atropia, then you have the diagnosis of iritis complete. I would recommend the use of atropia in all cases of inflamed eyes except where glaucoma exists, and one must always be on the lookout for glaucoma, especially in elderly people. Where there is any tension atropia should not be used, because if the pupil be dilated very much, the angle of filtration will be interfered with and the patient is liable to have secondary glaucoma following its use. This is so rare, however, I again say, that in all inflammations of the eye, except where glaucoma has been diagnosticated, I would make free use of sulphate of atropia. In cases of conjunctivitis where the cornea or iris is likely to be secondarily involved, it is better to use atropia for one or two days in order to guard against adhesions of the iris.

Before proceeding to the treatment I want to say a few words more about the serous variety of iritis, particularly to show the difference between this and the plastic and parenchymatous forms. A patient may have but little pain about the eye, simply complaining of blurred vision. Looking into the eye, the pupil, instead of being contracted and small, is found dilated, which may be deceiving. This frequently happens in serous iritis. Taking the patient into the dark room, by a close examination more or less sclero-corneal congestion will be found, although he complains of no pain worthy of mention; by taking a four-inch lens and throwing a strong light into the eye obliquely, or magnifying the different layers of the cornea, possibly only one or two little specks of lymph will be discovered on the posterior surface of the cornea, constituting what is known as the serous form of iritis. There is no plastic material thrown out into or on the iris itself. This form of inflammation is exceedingly dangerous on

account of the mildness of the symptoms and because the ciliary body is very liable to become involved, and we may have in this form of iritis a glaucoma following. Not long ago I treated a young lady twenty-three years of age with this form of iritis: glaucoma followed, and I had to go the other way and use eserine instead of atropia. In the majority of cases that I have treated of serous iritis there has also been present more or less plastic iritis, and when this occurs of course eserine cannot be used.

*Treatment.*—First and last in all cases, except where there is evidence of glaucoma, even after all symptoms of inflammation have subsided, use sulphate of atropia. The effect of this drug is double, or we may say treble. First it controls pain by its local anæsthetic effect; it contracts the blood-vessels of the iris, and in that way relieves congestion of the part and inflammation; by dilatation it keeps the pupil away from the anterior capsule of the lens, and thus prevents posterior synechia. In the acute stage of iritis I would use two grains of sulphate of atropia to one-half ounce of water; afterwards in weaker solution. It will be observed that I say after acute symptoms have subsided use weaker solutions of atropia, because otherwise we are not only liable to have the constitutional effects, but also the local poisonous effects upon the conjunctiva which will shut off the use of the mydriatic entirely. The conjunctivitis may at least be partially controlled or prevented by putting a small portion of sulphate of zinc in the mixture. I have seen several cases where the continued use of atropia was followed by conjunctivitis, and sulphate of zinc, one-sixteenth or one-twenty-fourth of a grain to one-half ounce of water, dropped into the eye occasionally, corrected the trouble.

As to the constitutional effects of atropia: This may be prevented to a large extent by pressing upon the tear-duct when the drop is put in, in that way preventing its running into the nose and throat. I have seen a great many patients who possessed an idiosyncrasy to atropia, a small fraction of a weak solution dropped into the eye causing a flushed face, dry throat, delirium, etc., so that they were laid up in bed for several days.

In the serous form of iritis a rheumatic element is usually present, or there is some disturbance of the alimentary tract or some fault of digestion, so that the physician must look after the constitutional treatment just as he would in the other forms of iritis. Put the patients upon tonics, as they are usually very much run down in gen-

eral health; give iodide of potassium or small doses of mercury, and in uncomplicated cases of serous iritis use the sulphate of atropia locally; bathe the eye in water as hot as it can be borne several times during the day; see that there is no increase of tension, and if a glaucoma supervenes it will be necessary to leave off atropia and use eserine. I have used pilocarpine with beneficial results in a great many such cases. I think eserine used as a myotic for any length of time is liable to produce a dry cornea or a dry conjunctiva, and to prevent this complication I have often used the following:

℞ Pilocarpine muriate, gr. i;
Eserine sulphate, gr. ½;
Aqua dest., ℥ss.

Or

℞ Pilocarpine (Alb), gr. i;
Eserine, gr. ½;
Olive oil (sterilized), ℥ss.

Instead of the sulphate of eserine I sometimes use the muriate or salicylate—one-eighth grain in about one-half ounce of water—with good effects. If eserine is employed it should be in weak solution and put in often, as strong solutions long continued are liable to produce plastic inflammation of the iris. In all forms of iritis look out for the cause, whether gonorrhœa, syphilis, malaria, or rheumatism, and correct it.

In the use of atropia my policy is to commence in the morning and drop a solution into the eye every few minutes, perhaps six or eight times during the first hour, then wait an hour or two and go through the same proceeding, the object being to get the rapid effect of the atropia. Sometimes where the iritis is acute I use a solution of six to ten grains of atropia to one-half ounce of water. I have used small crystals of atropia; but this is inadvisable, because we are more apt to get a constitutional effect from it. The indication is to get the pupil dilated as quickly as possible and keep it so. If pain is very great and the pupil obstinate, in addition to atropia I sometimes resort to local blood-letting. Of course, the indications for blood-letting depend entirely upon the condition of the patient. I have had very good results from artificial leeches. The best location for applying the instrument is just over the temporal bone. With the trephine you can make a round incision into the tissues; then by

means of a suction-pump, by gentle manipulation, you will find that the blood will escape freely into the cylinder. Better still is the natural leech when obtainable. In applying them, draw an imaginary line back from the canthus on the temple and put two to four leeches above that point and two to four below; the amount of leeching will, of course, depend upon the condition of the patient at the time; if the patient is anæmic, little blood should be drawn; if the condition justifies it, the leeches should be allowed to stay on until they drop off. Care should be taken not to apply the leeches too close to the eye; I have seen them get into the eye and do considerable damage. Occasionally the leeches will bite into an artery and severe hemorrhage will result, but this can usually be effectually controlled by firm pressure. After the leeches drop off, if the patient is in full blood, it may be advisable to bathe the parts in hot water to encourage free bleeding for a short time: if the patient is anæmic this should not be done. Atropia, where the iris is firmly adherent or the pain very great, will not break loose the adhesions. Leeches, hot-water baths, and morphia will be of much use then. Cocaine in five-per-cent. solution dropped into the eye is of much help. Hypodermic use of morphine is sometimes necessary to control pain until you can secure proper dilatation of the pupil by means of atropia. Bathe the eye with water as hot as it can be borne. Cold applications are generally not useful in iritis, especially in the serous form where there is a rheumatic element; with these patients, therefore, it is better to use hot water. When the pupil is well dilated you may feel pretty safe. You must look to the constitutional treatment; if the trouble is serous, dependent upon rheumatism or gout, treat these conditions according to the accepted teachings on that subject. Remember that the conditions most often associated with iritis are rheumatism, gout, gonorrhœa, and syphilis, and that the most frequent cause is syphilis.

# Laryngology.

## LARYNGEAL PAPILLOMATA; INFLUENZAL LARYNGITIS; LACUNAR TONSILLITIS, AND SOME OF ITS SEQUELÆ; RAILROAD ASTHMA.

LECTURE DELIVERED AT THE UNIVERSITY OF BERLIN.

BY PROFESSOR FRAENKEL,

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Gentlemen,—The little patient I bring before you first, this morning, is, as you see, a rather delicate, anæmic-looking child, seven years of age, who first came under our care about two years ago. She was then extremely hoarse and was suffering from some dyspnœa, so that her breathing was audible and somewhat wheezy. Examination with the laryngoscope showed the presence of a number of papillary tumors springing from the mucous membranes of the vocal cords.

We at once thought of removing them, but as the child was delicate, unused to laryngoscopic and mechanical manipulations, distrustful, nervous, and anxious, and the operation would be tedious and painful, we put her on the syrup of the iodide of iron, and had her come back every few days for practice in laryngoscopy and to make her grow more accustomed to manipulations and to the doctor. Under our eyes the tumors gradually disappeared as the child's general health improved, under the influence of the tonic, the fresh air, and the nutritious food we had prescribed. After three months not a sign of them was to be seen. The hoarseness had completely disappeared, the dyspnœa no longer existed, and everything had returned to the normal. She continued so for more than a year and a half, when the hoarseness began to show itself again. Once more the papillary tumors were to be seen. The child had in the mean time run down in

health somewhat, so that we put her on the syrup of the iodide of iron once more. So far its effect has not been marked this time, and I shall send the child around to allow you all to see the papillary neoplasm. She has become a most docile patient by this time, so that I am sure you will all, despite her age, be able to obtain a good view of the extremely interesting condition. You will find on the anterior third of the left cord a bullet-shaped papilloma, the size of a large pea; smaller ones are in the neighborhood.

The disappearance of the tumors before was due to a real reabsorption, gradual tissue retrogression (metamorphosis), that we were able to observe laryngoscopically. They were not loosened by convulsive coughing movements and coughed up. They were the result of a disposition in the child that was somehow dependent on her run-down condition. What exact constitutional connection there was between the general condition and the neoplastic tissue-formation would be hard to say.

The case is extremely interesting, however, owing to the fact that the development of papillary tumors of the air-passages in general seems dependent on a certain diathesis. They have a modicum of malignancy about them, since even after thorough and, it would seem, radical removal by operation they have a tendency to local relapse. They grow always from the laryngeal mucous membrane, and never from that of the pharynx or nose, and the prognosis as to return is never absolute, since, as we have said, local or at least regional relapse is very frequent.

In this case, despite nature's own cure of the case, we have the relapse. We shall try the syrup of the iodide for some time, and if the recession is not noticeable we shall remove them. It begins to look now as though operative measures would be necessary this time, since her general condition is not so markedly lowered as before, and we can scarcely expect so decidedly alterative an effect from the tonic treatment as would lead to tissue changes.

Gentlemen, this patient, a man of forty-nine years, came to us about a week ago because of hoarseness. This hoarseness was not always of the same intensity. At times he could speak scarcely above a whisper, at others he used a very high-pitched instead of his ordinary voice. The difficulty of phonation had come on rather suddenly after he had had what he calls a cold for some days. He felt out of sorts, had some fever, he thinks, lost his appetite, felt intensely tired,

had uncomfortable, almost painful, feelings in his bones, and a running from the nose. All this comes out under questioning after examining his throat, where, on the vocal cords, we find what we have cause to consider as characteristic changes.

The laryngeal mucous membrane is diffusely swollen and hyperæmic. The vocal cords are distinctly redder than normal, because of this inflammatory hyperæmia, and where the redness is most intense there occur whitish spots, which seem to be, however, on the same level as the surrounding hyperæmic parts. These spots occur especially to the front of the larynx, go to the middle, but not farther back. This is the characteristic laryngoscopic picture of influenzal laryngitis, as I described it during our first great epidemic of the disease here in Berlin. Not a year has passed since that I have not had a number of cases to treat, and the opportunity to exhibit some of the patients to the class. Influenza, though not in as severe a form as when it first spread so rapidly over Europe, has become, for the time being at least, endemic among us.

The lesions of influenzal laryngitis are so typical that the diagnosis may be made from them alone. The white spots seem to be fibrinous infiltrations of the mucous membrane and of the vocal cords, and when we consider the well-known tendency of the influenza bacillus to produce patches of fibrinous pneumonia in the lungs, it is not surprising to find these fibrinous lesions here. Their course is a rather chronic one. They remain almost in their original condition for two to three weeks, and after the hyperæmia of the cords has disappeared, they are still plainly to be seen.

After about fourteen days, and with the disappearance of the cordal redness, there is noticed around each of the spots a reddish areola, more or less distinct. Then the mucous membrane ceases to reflect light as before. The whitish spots become of a dirtier, darker shade, and with careful laryngoscopy we can detect slight differences of level, so that there is evidently loss of tissue, and manifestly this last stage is that of little ulcers. The spots take on a reddish color that forms as great a contrast with the now white normal vocal cords as the original white spots did with the hyperæmic red cords.

The affection, as I have said before, runs a chronic course in the first stage, and the closing stage also partakes of this chronicity. It is always six to eight weeks before the vocal cords have returned to their normal appearance, and it may be even longer. This extremely

chronic course is another sign that we have to do with a specific laryngitis utterly distinct from ordinary affections of the vocal cords.

It is not the only affection of the larynx that may develop during an attack of influenza. Influenzal hoarseness, for instance, may depend on a simple inflammatory hyperæmia of the vocal cords without the characteristic ulcers. The swelling of the laryngeal mucous membrane may extend to the subepiglottic tissues and present the series of folds beneath the cords that have been described as being the cause of pseudo-croup. Then the involvement of the muscles is not uncommon; especially does the infiltration affect the interni and transverse muscles. But these complications are common to all forms of laryngitis, while this spotty fibrinous infiltration of the vocal cords seems to be special to influenza in this region.

As to the treatment: Just as we have no specific for influenza itself in its principal manifestations, so there is none for this laryngeal complication. You know how slow and unsatisfactory resolution may be in an influenzal pneumonia, and the same thing holds here. General treatment, by making all systemic processes livelier, leads to more rapid resolution here as elsewhere. The patient must not be allowed to run down in his nutrition, and rest, as far as possible, of the vocal cords must be enjoined; especially must all unusual exertion, as singing or public speaking, be forbidden. Besides this the usual treatment of laryngitis by inhalations and the like will find a place.

Our next patient, a woman of forty, comes to us with the most common complaint that we hear of in a nose and throat dispensary,—a sore throat. Under the term sore throat, patients, however, understand practically all the affections that may occur in the pharynx, larynx, and on the tonsils, though, of course, it usually designates the most frequent of them, acute tonsillitis. As if to make up for patients grouping all the acute throat affections under one name, the doctors have any number of names for an ordinary angina, and the main difficulty in the study of the subject and the most fruitful source of confusion for the student, is the variety of names he finds in different authors for absolutely the same disease.

In this case the affection began three days ago, with a chill and fever. She had difficulty in swallowing, and a painful sense of constriction and dryness in the throat. She was in good health before, and has never had such an attack previously. All the symptoms con-

tinued to increase for three days, when the acme was reached, and to-day, the fourth day, she feels considerably better. That is a typical history of the affection, sudden onset, often with a chill, increase of symptoms for two or three days, then very often a sweat, and after that gradual relief, which sometimes, however, comes almost by crisis.

Patients usually attribute the disease to having caught cold, but this is the very natural human tendency to find a reason for every phenomenon. Careful inquiry will often show that there has been no special exposure to cold. There is no doubt, however, that exposure of the cutaneous surface to cold, with the consequent contraction of the capillaries of the skin, may act reflexly on the vasomotor, especially the vasoconstrictor, mechanism of the tonsillar capillaries, and so interfere with the protective processes that need to be so active in the tonsils, owing to the constant presence on their surface of the abundant pathogenic bacteria of the mouth.

The clinical picture as we see it here in the patient's mouth is an extremely simple one. The tonsils and adjacent tissues are red and swollen. The tonsils particularly project prominently into the mouth, and on their surface, which is more uneven than it usually is, there are points of white, which a little observation shows to be rounded, drop-like projections of a viscid, whitish substance. After the patient swallows the whitish points disappear for a time, or show only slightly and not so numerous as before, but if you watch the tonsils for a few minutes, they appear again and grow gradually larger. When they are removed by the act of swallowing, you can see that they were situated on the so-called crypts of the tonsils, the little infoldings of the superficial mucous membrane of the tonsil that occur all over that organ.

That there should have been any confusion in naming so distinctive a set of symptoms as these are is due to the fact that the physiology and anatomy of the tonsils have been in an unsettled condition during the evolution of the knowledge of its pathology and therapy, and all have shared the same fate in the confusion of terms that ensued.

The structure of the tonsil is not simple. Its groundwork is a mass of adenoid tissue, supported by a rich connective-tissue net-work. On its surface the mucous membrane dips down into the adenoid tissue, forming little pockets. These used to be called mucous follicles or the crypts of the tonsils, or the lacunæ tonsillares, lacunæ of

the tonsil. But as the name mucous follicle had already been appropriated to something in the intestinal tract, with quite a different function to that of these mucous pockets of the tonsils, the anatomical society saw fit to substitute for the word follicular the word fossulæ, meaning little excavations or hollows.

Meantime the name follicular tonsillitis was very generally known for the disease in which there was oozing of inflammatory material from the so-called follicles. This would, I suppose, now have to be called fossular tonsillitis. The affection is not, however, merely an inflammation of the mucous lining of the fossulæ; much more of the anatomical structure of the tonsil is involved than this.

In the midst of the adenoid tissue of the tonsil there occur two important anatomical structures, mucous glands and lymphatic nodules. The mucous glands are true acinous glands, situated in the substance of the tonsil, and with a duct opening on its surface. The noduli lymphatici, or lymph nodules, are collections of lymphatic tissue, situated all through the tonsil, but especially near its surface, from which there are always leucocytes wandering towards the periphery of the tonsil.

As the result of inflammation or inflammatory changes in the fluids of the part, white blood-corpuscles are attracted to the tonsil in greater abundance, and the leucocytes from the lymph-nodules wander out more plentifully. The result is, the approach to the surface of the tonsil of large numbers of cells, some of them already yellowish in color from fatty degeneration. The surface of the mucous membrane nearest the lymph-nodules is at the bottom of the crypts or fossulæ, so that most of the leucocytes find their way here, filling up the fossulæ, from whose orifices they project in the little, yellowish-white beads you can see so well here, until they are disturbed by swallowing movements, when for a time they are not visible. After a short interval they reappear, and the process of outwandering continues.

This abundance of leucocytes in this region has been by some thought to be connected with nature's protective effort at this point. The tonsils present a most favorable point for infection, being situated where they are constantly bathed by the secretions of the mouth, always so rich in bacteria of all kinds. Besides, for the purpose of physiological functions, all of which we do not as yet thoroughly understand, tonsillar tissues are loosely constructed, and so especially

open to the invasion of bacteria. Nature's special provision for their protection by the phagocytol action of out-wandering leucocytes is admirable, if Metschnikoff's theory of phagocytosis, as it would seem, represents one of the natural protective methods.

These leucocytes carry out with them the bacteria that have found their way into the tonsils, and it is their exit from the crypts or fossulæ that constitutes the distinctive feature of the disease. The disease itself is well known and easily recognized, but the variety of anatomical names that, for one reason or another, have been given to various anatomical features of the tonsil have given rise to a good deal of confusion as to its name. Sore throats are commonly called anginas, and as the affected part here, at least the one from which the puriform secretion was seen to issue, was what used to be called the follicles of the tonsils, the name angina follicularis had the advantage of being simple and descriptive,—follicular tonsillitis equally so.

But the so-called follicles of the tonsils, as we have said, do not at all correspond to the anatomical organs to which the name follicle has been given throughout the rest of the intestinal tract. By the name follicle was meant not the lymphatic nodules of the tonsils, but the little crypts or pockets in the tonsil, where the mucous membrane covering them dips down into the body of the tonsil.

Wagner, of Leipzig, then suggested the name angina lacunaris, what may be called lacunar tonsillitis, the word lacuna meaning a little hollow, a crypt. This term was descriptive, and was not open to the objection of misrepresenting the anatomical arrangement that was supposed to be at the bottom of the disease. The anatomical society, at its meeting for the adoption of anatomical terms that would put an end to the confusion that had arisen from the careless use of terms and their use by different schools for different objects, added another element to the confusion of names already existing here for the laryngologists. They selected as the name for the crypts of the tonsils, not follicula and not lacunæ, but fossulæ, a diminutive of fossa, a ditch.

If we should follow the old idea and give a descriptive term to the disease, it would be fossular tonsillitis or angina fossularis. As the real origin of the pathological secretion seen in the throat, however, is not the crypts of the tonsils, but the lymphatic nodules in the body of the tonsil, the proper name, because stating the pathogeny and

pathology of the disease, would be angina nodularis, nodular angina or tonsillitis. You see the number of names there are and what confusion they give rise to in the literature. I beg of you, then, when you describe a new throat disease, as all of you doubtless will when you have become distinguished laryngologists, do not be overhasty in giving it a final name; wait until further study has enabled your contemporaries to help in the always difficult task of selecting a name for the new-born. Meantime I may say that I suppose the name suggested by Wagner, angina lacunaris, is at once readily understood, and does not easily lend itself to misinterpretation.

So much for our digression into the nomenclature of the commonest of throat diseases. Now what are we to do for our patient? I have already asked her, and been assured that she is much more comfortable this morning. As so often happens after two or three days of the disease, there was a sweat last night, followed by a fall in temperature, a recovery from the feeling of general discomfort that had preceded, and then a beginning disappearance of the local symptoms, pain and swelling. In nearly every case the disease follows this cyclic course, and gets better by a sort of crisis. All manner of remedies have been recommended and have seemed to do good, since their administration has been followed by relief of the symptoms. As to whether this relief was consequent or only subsequent to the treatment is not considered. It was the same way in the old days with regard to internal remedies for pneumonia; it is to a certain extent true for erysipelas in our day, though its self-limitation is coming to be more generally recognized.

For the specific treatment of lacunar tonsillitis there is none, nor is any needed, since the disease rarely runs a dangerous course. Symptomatic treatment, especially for the sore throat, may be employed with advantage. Cold drinks and the swallowing of small pieces of ice usually relieve the pain, though sometimes warm applications are more welcome. Chlorate of potassium still remains a favorite remedy for many, and is perhaps of benefit. A five-per-cent. solution, as a gargle, three or four times a day, may be employed. There is no necessity to reduce the patient's food. Children especially should, if possible, be induced to take plenteous nourishment, of course soft and cold, if it is more pleasant, for convalescence is then much more rapid when it sets in, if the patients are not weakened by having eaten but little for some days.

The rounded, beady appearance of the secretion that wells up at the entrance to the fossulæ of the tonsils is very characteristic for the form of angina that we have been speaking of; it is to be remembered, however, that there occur cases of diphtheritic angina, in which the membrane appears on the tonsils in spots. These are, however, persistent, do not change their appearance after the patient has swallowed, to recur some time after when more secretion has made its exit from the fossulæ. The confusion of these two forms has led to the reporting, at times, of paralysis after mistakenly called lacunar or follicular tonsillitis, which was really diphtheria. Sometimes, however, the appearances are so dubious that only a bacteriological investigation will decide the question of etiology.

The sequelæ that may be expected sometimes to follow lacunar tonsillitis are purulent peritonsillitis, peritonsillar abscess, and the tendency of the disease to recur after it has once affected the tonsils. There is no prophylaxis that will enable us to prevent peritonsillar abscess; it is likely in many cases that the tonsillitis is but the first stage of the acute inflammatory process that is at work deeper in the tissues. The treatment of it is to open it. I show you here the patient whose peritonsillar abscess we opened yesterday. There is still some pus issuing from the opening, but the symptoms are so far in abeyance that the patient suffers scarcely any discomfort from what yesterday was so unbearably bothersome.

As to the tendency to recurrence. The only remedy for it is the removal or thorough cauterization of the tonsil. In children, in young people generally, a simple tonsillotomy is the easiest way. In general, where the tonsillar tissues are hard, showing an extensive overgrowth of connective tissue in the tonsil, from repeated acute or persistent chronic inflammation, then it is not advisable to remove the tonsil by cutting, owing to the danger of hemorrhage from the section, which is sometimes extremely hard to control. The overgrowth of connective tissue seems not to allow the cut vessels to contract properly, hence the hemorrhage.

Where the patient is older or where the tonsils are hard to the feel, then the electrocautery, used in several sittings, enables us to attain practically the same result. The thorough searing of the surface of the tonsil seems to prevent recurrence of the tonsillitis quite as well as the extirpation. Of course, the so-called extirpation is so only in name. We do not remove the whole tonsil, but only amputate the

part of it that projects beyond the pillars of the fauces, and the operation should be called amputation of the tonsils. Sometimes, even after this operation, the tonsillitis recurs, but it is extremely rare. I have never seen any of the reflex sequelæ that have sometimes been spoken of as occurring after tonsillectomy, in particular. I have never seen any change in the genital apparatus, or any sign of the atrophy of testes or ovaries that has been at times said to have occurred.

Here we have a patient who is suffering from her fourth attack of tonsillitis in two years. Evidently this is an appropriate subject for radical treatment of the altered condition induced by inflammatory infiltrate, which has made the tonsillar tissues less resistant to infection. She is a girl of eighteen, but her tonsils are not hard from a chronic inflammatory process, so that, I think, we are perfectly justified in doing a simple tonsillotomy. In her case it is all the more indicated as she herself does not think that her circumstances will permit her to stay in the hospital the length of time that would be required for thorough cauterization. I simply remove her tonsils with the tonsillotome. She shall be the subject of special attention to-day as regards bleeding. In three days she may leave the hospital.

Our next patient, gentlemen, is a man of forty years, whose family history is good, and whose personal history has in it nothing of importance for us. He comes because of a special set of symptoms that obtrude themselves, whenever—in the summer time especially—he rides for long distances on a railroad train. He first has ticklish sensations in his nose, then he begins to sneeze, and finally convulsive sneezing sets in, with but short intervals. At the same time there comes a sense of compression and difficulty of breathing through the nostrils, the left nostril closing up entirely. There is a tendency to increased flow of tears and some reddening of the conjunctiva. Shortly after leaving the train the symptoms subside.

The products of combustion from the engine seem to have nothing to do with the etiology of the condition, as it occurs in his case whether he rides at the end of a long train or immediately after the engine. In winter it does not occur, and is but slight in warmer weather if the day is damp or rainy, so that the cause would seem to be the dust raised by the train in its passage, which, while the windows are open in the summer time, finds ready access to the cars.

We have here, gentlemen, one of those rather rare cases of what

I described some years ago as railroad catarrh, or asthma; the English speak of it as hay asthma or railroad asthma. It has, indeed, many analogies with that much-discussed disease, and, in fact, occurs in some people who are liable also to attacks of hay asthma. People affected by both say that the beginning and course of railroad asthma is very like the other, and produces practically the same set of subjective and objective symptoms.

An examination of the nose of our patient shows that there is some hyperæmia and some increase in size, though one could scarcely call it hypertrophy of the turbinated bones. This enlargement is not localized, but affects all the visible surface of the turbinates. Some such pathological condition as this is usually found in the nose of people who suffer from railroad catarrh. Sometimes there is a hypertrophy of the tuberculum septi; sometimes the existence of an eczema at the entrance to the nostrils seems to have a causal relation to it. A good many other anomalies in the nose have been thought to account for it; deviations of the septum, polyps, ulcers, and the like. Occasionally, however, people are found who suffer from the disease, and yet have absolutely no pathological condition in their noses at all.

As the affection seems to be due entirely to the presence of particles of dust, it would seem that we must conclude that there is in these cases a supersensitiveness of the nasal mucous membrane. This hyperæsthesia may be the reflex result of some pathological obstruction of the nose, but it may be due to reflex hyperæmia of the nasal mucous membrane from quite other causes. The irritation of the hyperæsthetic sensory nerves by fine particles of dust sets up motor and vasomotor reflexes, hence the convulsive sneezing, the dyspnœa, the swelling of the mucous membrane, rendering breathing through the nose impossible, and the increased secretion.

As regards the treatment of the condition. Where we find evident pathological conditions present in the nose, these must, of course, be overcome as far as possible. Eczema introitus nasi, and hyperæmia of the tuberculum septi must be treated on the general principles suitable for these conditions. Deviations of the septum must be corrected, polyps and localized hypertrophies of the turbinated bones must be removed.

For the hyperæsthetic irritability of the nasal mucous membrane and for the indefinite intranasal condition that are found in cases like

the one we have here under consideration, I consider injections of weak solutions of nitrate of silver the best remedy we have. Galvano-cauterization has become so handy and so painless in our day that it is abused, especially in cases like this. It has its place and a most useful one, but that is practically limited to the cauterization of definitely localized lesions. For the general irritability of the nasal mucous membrane nitrate of silver is much more advisable.

I begin the treatment with a one-fourth per mille solution of nitrate of silver, gradually increase the strength of the solution to one-half per mille or higher, but never use a stronger solution than one per mille. I inject a single nasal syringeful and neutralize the superfluous nitrate of silver with a second syringeful of physiological salt solution. The injections I always make personally, and never intrust them to the patient himself. They may be made every day, though I prefer to make them not oftener than once every second day. Some patients have an idiosyncrasy for nitrate of silver, and its use in the nose even in very dilute solutions is sometimes followed by migraine-like attacks that in many cases are very severe. The injections should, therefore, always be begun with the greatest precautions, and a very weak solution. Where even with the weakest solution the migraine symptoms still occur, the injections must be given up.

As a prophylactic against the attacks, some people find the putting of sterile cotton tampons, dipped in a little vaseline or lanoline, in the external nostrils of use; other patients find these so uncomfortable as to be unbearable, and for some the added irritation of this foreign body in the nose seems to increase the severity of an attack.

# Dermatology.

## PRURIGO; INDURATED SORE OF LOWER LIP IN A CORNETIST; PITYRIASIS ROSEA; INJURIES, IRRITATION, AND LOCALIZATION OF SYPHILITIC LESIONS.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF BERLIN.

BY PROFESSOR LASSAR,

Professor of Skin and Venereal Diseases at the University of Berlin, Germany.

Gentlemen,—I have a series of very practical cases to show you this morning, that will be of special interest to most of you, since the greater number of you are preparing rather for general practice than to make a specialty of skin diseases. These cases are just the sort that come to the general practitioner first, as they have neither the unsightliness nor the urgency, nor seemingly the seriousness, that would tempt patients to seek a skin specialist's advice.

Our first patient this morning is a little girl of six, who has already been under a general practitioner's care for some time, and, as the disease failed to yield to treatment, he has referred her to us. Her mother says that her present condition has developed mainly during the last month or two, though she admits, too, on being carefully questioned, that during preceding summers the child has had something of the same kind, only not so bad. In fact, she cannot say definitely when she is sure that she saw the first signs of the affection, though she does not think there was any sign of it when the child was very young. The history is a rather typical one of the condition, and so deserves your special attention.

The most noticeable thing about the cutaneous picture presented is the presence of scratch-marks. There exist on the extensor surface

of the arms here, especially the forearms, and on the thighs a series of punctate lesions whose original appearance is obscured by having been scratched. They are now red and irritated-looking, many of them covered at the point with small scabs where the epidermis was removed. The form of the lesions, their itchiness, and their position here on the extensor surface of the forearm make us think at once of scabies. It was for this that she was treated before coming here. But there are no lesions between the fingers, no burrows and no parasites are to be found, and the ordinary treatment for scabies usually so effective has had no effect in this case. It is worthy of note, too, that the genital region and the triangle just above it are free from lesions, though they are so common here in scabies, infection easily taking place from the hands, where the parasite usually begins its attack.

With a history of indefinite duration, beginning, however, a good while ago, and probably existing for years, we think of another disease that gives rise to persistent itching,—prurigo. Some of the lesions here on the trunk are still in their original state, undisturbed by the scratching. They are minute papules of the color of the normal skin, but very slightly elevated above the general level of the cutaneous tissues and rather to be felt than to be seen. The first lesions of the disease occurred on the legs, at least it was the child's scratching here that first called the mother's attention to the affection. This is the most frequent location for the first lesions to appear. There are some lesions here on the face also that have been scratched, and this has been followed by an eczematous condition, that would still further obscure the diagnosis unless all the circumstances of the case were taken into account.

This complication of prurigo with other diseases is not infrequent, and leads to mistakes of diagnosis. Some time ago I had a Russian to treat who had been bothered for years by an intractable, persistently relapsing, facial eczema, careful questioning of whom brought out the fact that for years he had had a slight but distinct prurigo. It was this that had formed the groundwork for the persistently recurring eczema. In my opinion, the lesions of prurigo occur on the face oftener than is usually said to be the case, the spots being but few and escaping notice when the eruption on other parts is so striking.

Prurigo, I think, often exists for years without being suspected. People are itchy and uncomfortable at certain seasons of the year,

but they attribute it to the heat or the cold, as the case may be, and think no more of it. In such cases, when for some special reason the disease becomes severer and they consult a physician, it is not always easy to get a history of preceding attacks of the disease. It is not an unusual thing to see people who squirm a good deal, whose clothes seem to irritate them. Many of these cases are really subjects of prurigo in its lighter forms, and now it is always my custom, when I get an eczema absolutely localized to the face, for instance, to ask patients about squirming. An objective sign of prurigo having been present is sometimes seen in the enlarged glands that occur as a consequence of slight infections of the scratch-wounds.

Of the definite etiology of the disease we do not know very much. It is a disease of the poor, that begins at a very early age and continues for years unless carefully treated, and then is liable to frequent relapses. It is usually worse in winter, though sometimes, as you see in this case, the first serious symptoms occur in summer.

While the prognosis as to lasting cure is not very favorable, as can be gathered from what I have just said, yet treatment is of the greatest service, and sometimes in the young leads to the complete disappearance of the symptoms. Of course the first indication is to relieve the itching and so preclude the occurrence of further scratch-lesions. When the patient is in an intensely irritated condition, as here, soothing remedies are required, and we shall first prescribe an ordinary dusting-powder of oxide of zinc and starch. Often in these cases you will find that the parents have tied the hands of the children in order to keep them from scratching. This makes the condition a veritable torture, for nature's itchiness demands some relief by the counterirritation of scratching. So it is better to leave the child's hands free, but thoroughly cover the affected parts with bandages, through which a certain amount of rubbing may be permitted that will not produce additional lesions.

As soon as the irritation has subsided, the treatment proper of the prurigo will be begun. We use here Wilkinson's ointment, but make it milder by the addition of a domestic soap, instead of sapo viridis, and increase the amount of vaseline:

℞ Ol. Rusci, 6. (℥iss);
Sulph. sublim., 6. (℥iss);
Sapo domestic. pulv., 12. (℥iij);
Vasel. flavi, 12. (℥iij);
Cretæ albæ, 4. (℥j).

This for milder cases or where the patients have sensitive skins. For severer cases:

℞ Picis liq.,
Ol. Rusci,
Ol. Fagi, āā 40. (*i.e.* about ℥i);
Spir. dilut.,
Ol. olivæ, āā 10. (*i e.* about ʒij).

Whichever one of these is selected is applied just before putting the patient into a bath, and the patient is left in the water from half an hour to an hour. After the bath a layer of brown salve is employed, and over it, as a varnish:

℞ Zin. oxid., 60. (℥ij);
Ol. oliv., 40. (℥i).

Some indifferent powder is spread over all, and then the patient is carefully bandaged. The bandages are left on for three days, when the treatment is repeated. In an inveterate case it may have to be repeated several times. Usually at the end of three weeks the patient is well.

If there is constipation, compound licorice powder is the most satisfactory laxative.

## INDURATED SORE OF LOWER LIP IN A CORNETIST.

Our next patient is a man of forty-three, a cornet-player, who noticed about six weeks ago a sore on his lower lip. This has persisted in spite of all the remedies he has used for it. He has not been able entirely to give up his occupation, because he and his family are dependent on his wages for their living; but he has of late limited it as much as possible, yet still the sore refuses to heal. He is naturally somewhat worried about it, and has come for a definite opinion in the matter.

We find here on the lower lip, slightly to the right of the median line, a lesion the size of a five-pfennig piece (silver five-cent piece). It occupies especially the line of contact between the mucous membrane and the skin, though extending farther on to the mucous surface. It is covered by a thin crust or scab, and is but very slightly if at all raised above surrounding tissues, though its edges are sharply defined. To the touch it is only of medium hardness, and has certainly not the induration that is usually found in either cancroid or

chancre in this region. The submaxillary lymphatic glands are not enlarged, and, according to the history, the lesion has shown no special tendency to spread.

We naturally think of the two affections which I have just mentioned,—cancroid, or epithelioma, and chancre, *i.e.*, the initial lesion of syphilis. Owing to the absence of characteristic induration, though there is some induration of the sore, another disease, comparatively mild and transient, as a rule, which the constant irritation of his occupation might have made chronic and indurative, must be considered in the differential diagnosis.—namely, herpes.

Against herpes speaks the duration of the affection, six weeks, but the persistency may be due to the persistent irritation. Against herpes also is the fact that this is the first time he has ever had such a sore, and that the lesion was from the beginning single. For herpes, or some simple, non-specific, benignant process, speaks the comparatively slight induration that has been produced, though the affection has now lasted over six weeks, the absence of glandular involvement, and the fact that it began as a small bleb.

Against chancre there are the same factors,—the absence of enlarged glands and the presence of but inconsiderable induration. For it speak, to a certain extent at least, the chronicity of the affection, the practical absence of anything like destructive ulceration, and the bit of anamnesis which he has volunteered,—that about ten days before he first noticed the sore he used another cornetist's instrument. He has thought of possible infection in this way himself, though he confesses that the other man is perfectly healthy to all appearance, has never to his knowledge been troubled by a skin eruption, and is a married man whose wife and children are healthy. Our patient himself has never had syphilis. We cannot, then, exclude the possibility of the lesion being a chancre.

Finally, there is the third possibility, the one which in a patient of this age and a lesion situated where this is, on the margin between skin and mucous membrane, must ever be a doctor's first thought. Is it labial epithelioma? The chronic course, its occurrence in a man of forty-three, whose occupation subjects his lips to a great deal of irritation, who is besides a heavy smoker,—all these make the lesion a very suspicious one in this regard. We could scarcely expect epitheliomatous glandular involvement as soon as this. But against this diagnosis of cancroid, or labial epithelioma, there is the absence of

sufficient induration to justify the thought that the exudation here is an epithelial neoplasm.

We cannot, however, make an absolutely definite diagnosis in the case; the lesion may, as a matter of fact, be any one of the three affections whose differential diagnosis we have discussed. It may be a patch of chronic inflammatory exudate on the basis of a lesion of herpes, its chronicity being due to the irritation incident upon the pressure of the mouthpiece of the cornet and the effort of blowing the instrument. It may be the initial lesion of syphilis acquired from the use of another's instrument, a not unusual way of acquiring syphilis, as you know, and one that must be constantly looked for in the history of patients who are liable to be placed in circumstances in which they may be tempted to use something that others have had in their mouths. The blowers of wind instruments, glass-blowers, and people who borrow pipes have furnished a number of examples of extragenital chancres for medical literature.

It may still be chancre even though not acquired from his friend's instrument; for people who use their lips as much as he does often have excoriations, especially in inclement weather, and these may become infected from other sources,—drinking-cups, beer-glasses, knives and forks, and the like. Abrasions of the skin or mucous membranes often furnish the occasion for inoculation, though their occurrence may not have been accompanied by actual infection. Chancres that occur in razor cuts, for instance, may not have been directly inoculated from the razor itself, which is not difficult to clean, and so must be an infrequent carrier of infection, but may have absorbed infectious material from the hands or towels of the barber, which are much more likely sources of infection.

Finally, we must not lose sight for a moment of the possibility of this being, at our patient's age, epithelioma, for it is just as such innocent-looking sores on the lip that they originate. Neglect of them is, of course, serious; if we should wait until the glands are enlarged, it is doubtful whether we should be able to eradicate all the cancerous elements by operation. We shall give our patient, then, a full course of specific treatment by mercurial inunctions. If after three weeks there is no involution noticeable in the sore and it does not react favorably to the local treatment, we shall counsel its excision. This will be comparatively simple, and will in this situation cause practically no deformity, the labial tissues readily stretching

to make up for the defect produced. At his age neglect of a lesion like this is a serious matter, and if you allow yourselves to put off too long the suggestion of operative interference you will regret it.

## PITYRIASIS ROSEA.

This patient is a man of thirty-five, who first noted some itchy red spots on his buttocks about three weeks ago; since then they have spread all over his body, as you see. These spots are for the most part not raised above the surrounding skin, and most of them are not spots in the sense of macules, but are circular lesions whose centre has healed. The healing process, however, led to some pigmentation, and so there is a characteristic brownish color of the centre of the lesions that is almost pathognomonic of the disease. Besides the eruptive spots are covered with fine scales. We evidently have to do with the skin affection known as pityriasis rosea.

The patient says, when questioned more closely, that he first noticed the eruption here on the pectoral folds at the front of his axillæ and in the axillæ themselves, then on his buttocks, but, as the eruption was already rather extended when he noticed it, it may have existed here first. It is now pretty generally and regularly scattered over his trunk, being especially plentiful at the parts that are most liable to be irritated by folds of his clothes and by other external agencies,—in the axillæ, over the buttocks, around his waist, at the nape of his neck, etc. He did not wear a new shirt, and, he thinks, was not wearing one that had not been worn for some time, just before the eruption occurred. I have asked him these two questions because the wearing of such a garment often constitutes the only etiological factor that we can find for the disease. He was also not constipated just before the eruption, had eaten no unusual food so far as he remembers, and had no symptoms of indigestion, so that we would seem to be able to exclude the autotoxic hypothesis that some have advanced in explanation of its etiology.

The important thing about the disease is that we can assure the patient that it is not serious and that it will be well in a couple of weeks at most. It usually runs a definite course of from two to five weeks, and, as it has already existed for some three weeks and the state of involution of these plaques here on the thorax shows that it has begun to disappear, I think that our assurance will not be belied by the result.

An examination of some of these spots on different parts of the body will show the course of the lesions. They begin as small pinkish-red spots, that coalesce with neighboring ones, show signs of some flaking, are redder at the periphery, where they are advancing, and gradually disappear in the centre, a little fawn-colored patch appearing here. Gradually the whole erythematous lesion disappears, leaving only the fawn-colored patch.

As to treatment very little is needed beyond the assurance to the patient that the disease is perfectly innocent and that it will surely get well without long delay. There are certain remedies which seem to hasten its course somewhat. Of these tar and sulphur have given me the most satisfaction. I either give a mild tar ointment or have the patient use tar soap freely and then take a bath in sulphur water. Of course, if there is constipation or some other systemic disturbance, that should be treated, for its continuance may retard the involution of the disease. It is under such circumstances that the few longer-drawn-out cases that have been reported have occurred. The disease is much more frequent in children than in adults, though our patient is an adult, and in children particularly, when the patient gets warm in bed or is sweating, the lesions become itchy, and this condition will require treatment with sodium hyposulphite or mild carbolic lotion.

## INJURIES, IRRITATION, AND LOCALIZATION OF SYPHILITIC LESIONS.

We have next, gentlemen, a set of three cases of the old, old story, that must ever be new and interesting to any one interested in dermatology. All three of these patients confess to having had syphilis, so that the diagnosis is easy, and it is the origin and localization of the lesions that attract our attention.

The first is a waiter in a restaurant, who had his initial lesion seven years ago, and who has been under treatment at several doctors' hands since. He fully realizes the nature of the disease and has carefully followed prescriptions given him, so that the frequently recurring relapses and the appearance every now and then of lesions of so-called tertiary syphilis cannot be laid to therapeutic neglect.

He has been under treatment several times in the last three years in our out-patient department, usually for cutaneous or subcutaneous

gummata. Once before it was a gumma of the arm following a blow. Now there is a tumor on the proximal phalanx of his thumb, just where in the pursuit of his occupation as a waiter he has his thumb pressed on the edges of plates and trays in order to carry them. At times, of course, plates that he handles are hot, and this frequent overheating of the tissues at this point is probably another reason for the lowered resistance of the part and the consequent localization of the lesion here; for it is well known that gummata occur by preference in tissues whose vital resistance has been lowered by trauma or by some other extraneous cause. There are even those who hold that the occurrence of a gumma in the internal organs is always preceded by some pathological condition in them.

Another case of the localization of gumma because of trauma we have in this second patient. He had syphilis about four years ago, was thoroughly treated, and has had no symptoms for nearly three years. About four weeks ago he received a kick on the shin. A sore spot developed that was extremely slow to heal and that finally showed a tendency to break down. It was treated first by home remedies, salves and ointments, and afterwards by a physician, who, however, knew nothing of his having had syphilis. As the sore got worse instead of better, he came here, where he had been treated for his syphilis and where our knowledge of his history enabled us to make the diagnosis of cutaneous gumma in an ulcerative stage.

But not only the tertiary lesions occur where trauma or irritation has furnished a suitable nidus for the location of the virus, but also the secondary lesions localize themselves in a similar way, or at least occur earliest and most plentifully on parts where there has been irritation. I need only remind you of the collar of Venus which usually occurs in women, whose neck-wear is much more irritating than that of men, while the crown of Venus, the series of lesions that occurs so often on the forehead at the margin of the hair, is seen oftenest in men, their head-gear being a source of special irritation from heat and pressure.

Here we have a case where trauma of the scalp has been followed by loss of hair. The patient bumped his head severely about three weeks ago, the injury was followed, as he says, by a lump that has gradually subsided, but now where the lump was the hair is coming out. The fall of hair is in the patchy way so characteristic of syphilis, giving a piebald appearance at the spot, and, if the bald areas be

examined a little more closely, each of them will be found to be the site of an erythematous papular eruption.

These three cases are interesting because the history of injury and exposure to irritants in them might have distracted attention from the underlying condition, to which their characteristic development in some and their chronicity and obstinacy to treatment in all three of the cases are really due. This might easily lead to the employment of only local measures for their relief. It is always to be remembered, then, first, when a patient with a syphilitic history acquires traumatic lesions that do not correspond to the force of the trauma, but are followed by exaggerated symptoms, and when simple bruises and wounds fail to heal promptly, that probably the underlying constitutional diathesis is an additional etiological factor in the case, and indeed the one that requires prompt and effective treatment if the condition is to be improved; secondly, where a syphilitic history is denied or is doubtful, and simple traumatic lesions seem to give rise to chronic exudations simulating neoplastic formations that are obstinate to treatment, the case should be considered as suspicious, and, after a reasonable trial for a week or two of ordinary measures, if no amelioration occurs the therapeutic test of specific treatment should be given without further delay.

An interesting question that naturally occurs in connection with these cases of syphilitic lesions following trauma is the possibility of making latent syphilis artificially manifest. It is a question that occupied the physicians of a former generation much more than ours, but has not lost its interest for us. The idea, of course, is to judge of the advisability or non-advisability of another course of specific treatment for syphilis when for a good while it has been absolutely latent. If an injury is followed by syphilitic manifestations, the necessity for further treatment is evident; but, on the other hand, if a slight injury, an abrasion, a burn, or the like, is not followed by any local syphilitic symptoms, can we assume that the virus of the disease is not in a condition to produce any lesion in case of favorable circumstances presenting themselves?

Physicians used to think that they could determine the virulency of the syphilitic process in the individual at any given time by his reaction to certain remedies. For instance, the waters at Aix (Aachen) enjoyed the reputation of bringing out the symptoms of latent syphilis if it was at all virulent. The idea probably originated

with the doctors, but, when they had come to recognize the futility of the claim, the laity still clung to it, and syphilitics who took a course of the waters at Aix without the recurrence of symptoms were the subjects of congratulations from their friends who were in the secret. Some years ago a Russian surgeon announced that the use of certain caustics would be followed by syphilitic manifestations in the cauterized part, that would be easily recognizable as specific, if the syphilitic virus were only dormant in the system but liable to produce symptoms if the occasion presented itself. Further observation, however, showed that, even where cauterization had given negative results, subsequent injuries, even slight, especially such as involved the central nervous system, were followed by syphilitic manifestations. Not long ago a member of the Berlin Medical Society drew an interested audience of medical men to hear of "A method of making latent syphilis manifest," but it was only to discuss the method with cauterization which I have just spoken of, and to show that it was not trustworthy. Though the audience was disappointed, the speaker attained his object of attracting attention.

There is no way of being definitely sure that the syphilitic virus has been gotten out of the system. When it manifests itself, treatment must be given once more. Cases turn up, like our first case here, where lesions occur off and on for years; others where, after years of absolute latency, without warning almost, the most serious symptoms develop. The first course of specific treatment must be a thorough one, and must be continued with proper intermissions for at least two years. For many cases that will put an end to all symptoms. Not only, however, is the disease symptomatically cured, but in many cases it is actually at an end, as much at an end as any of the infectious fevers when their symptoms have disappeared. The best proof, to my mind, of this absolute curability of syphilis is the occurrence of infection a second time. This does not happen often, just as second attacks of smallpox are not frequent, but there are now in the literature too many cases of second infection for us to have any doubt about it.

To my mind, the very rarity of these cases shows, too, what seems to be well proved from other considerations, that one attack of syphilis usually protects from further infection, and only in very infrequent cases—at least as infrequent as smallpox, for instance—does the protection run out. After once having had syphilis people are

much more prone to be careless in exposing themselves, yet very few cases of second infections with syphilis have been reported. It is evident, then, that there must be a large number of completely cured cases of syphilis in which the protective immunity remains perfect. This natural resistance to secondary infection argues that many more cases of syphilis are completely cured than is usually thought to be the case.

I do not think, then, that we should, as some physicians advise, have our patients take a course of specific treatment every year for the rest of their lives. This is unnecessary in many cases and may be harmful. It smacks somewhat, too, of metallotherapy, the metal being precious and being absorbed by the doctor, not by the patient. When symptoms occur, of course they should be promptly treated, and after their original course of specific treatment patients should be warned of the liability of relapse, and should be enjoined to let no symptom —however simple or unimportant it might seem, if at all obstinate to treatment—run on without a physician's care or without informing the doctor of the previous syphilitic infection.

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